ATLAS

OF

NEUROPATHOLOGY

PREPARED AT

THE ARMY MEDICAL MUSEUM

OFFICE OF THE SURGEON GENERAL

U. S. ARMY

ATLAS OF NEUROPATHOLOGY

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THE ARMY INSTITUTE OF PATHOLOGY

OF THE ARMY MEDICAL MUSEUM

OFFICE OF THE SURGEON GENERAL

WASHINGTON, D. C.

By

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1944

PREFACE

The American Psychiatric Association through its Committee on Psychiatry in Medical Education takes great pleasure in presenting to the Army Medical Museum and its Curator, Colonel J. E. Ash, a collection of neuropathologic slides to constitute a central registry of neuropathology. The history of the development of such a slide collection for review in both graduate and postgraduate education is interesting. The organization of registries under the auspices of the Army Medical Museum is as follows:

1. Ophthalmic Pathology - Am. Acad. O & O - 1922
2. Lymphatic Tumors - Am. Ass'n. Path. & Bact. - 1925
3. Bladder Tumors - Am. Urol. Ass'n. - 1927
4. Dental & Oral Pathology - Am. Dent. Ass'n - 1933
5. Otolaryngic Pathology - Am. Acad. O & O - 1935
6. Dermal Pathology - Am. Acad. Derm. & Syph. - 1937
7. General Tumors - Am. Soc. Clin. Path. - 1937
8. Kidney Tumors - Am. Urol. Ass'n. - 1938
9. Lung & Chest Tumors - Am. Soc. Thorac. Surg. - 1940
10. Prostatic Tumors - Am. Urol. Ass'n. - 1943
11. Orthopedic Pathology - Am. Acad. Orthop. Surg. - 1943

The completion of this work was made possible through a grant to the American Psychiatric Association by the Rockefeller Foundation. It is a pleasing obligation to pay tribute to Dr. Karl Neubuerger who selected and prepared the major portion of this slide collection in cooperation with other outstanding neuropathologists in America. The collaboration of the American Psychiatric Association and the American Neurological Association and other interested special societies and individuals in the completion of this project is evidence of the essential close relationships between allied specialties. From the view point of our educational objectives we hope this slide collection and central registry of neuropathology will be used frequently

by interested members of the American Psychiatric Association especially younger state-hospital physicians, and others who do not now have the advantage of neuropathologic material for study. Our committee likewise hopes that this initial nucleus for education will grow and expand in its usefulness and availability to all physicians in the specialty of psychiatry and neurology.

Arthur H. Ruggles

Arthur H.Ruggles, President
American Psychiatric Association

Franklin G. Ebaugh

Franklin G. Ebaugh, M.D. Chairman
Committee on Psychiatry in
Medical Education

John C. Whitehorn

John C.Whitehorn, M.D.

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Baldwin L. Keyes, M.D.

Harry A. Steckel

Harry A. Steckel, M.D.

John Romano

John Romano, M.D.

FOREWORD

This is a first attempt to build up a loan slide collection in neuropathology. It contains slides of most of the important and frequent changes in the central nervous system. Some slides are not entirely satisfactory and should probably be replaced. The collection of slides on the pathology of the peripheral nerves and of the spinal cord is still incomplete, since appropriate material was not available. A number of rarer brain conditions is missing, such as: encephalitis in Rocky Mountain spotted fever; Wilson's disease; Parkinson's disease; gumma; status marmoratus; brain changes in some forms of idiocy; lobar sclerosis; cerebellar atrophies, and others. Furthermore, some unusual forms of brain tumors are not contained in this collection. Finally, additional slides with special stains may be desirable.

It will be greatly appreciated if every borrower of this collection will endeavor to furnish valuable material, for the completion of the set, and make constructive criticisms.

The friendly co-operation of Dr. Walter Freeman, Washington, D. C., Dr. George B. Hassin and Dr. Arthur Weil, Chicago, and of Dr. Helena Riggs, Philadelphia, in building up this set is acknowledged.

Karl T. Neubuerger, M.D.

University of Colorado School of Medicine

NOTE

This syllabus to accompany the loan sets was prepared at the Army Medical Museum, Office of the Surgeon General, U.S. Army, by the painstaking efforts of Major W. W. Hurteau, Medical Corps, U.S.A., and Captain Webb Haymaker, Medical Corps, A.U.S. with the cordial cooperation of Dr. Neubuerger. The sets as originally prepared were critically reviewed with the aid of several consultants, especially Dr. Walter Freeman of Washington, D. C., resulting in substitutions and deletions that have corrected some of the deficiencies mentioned in Dr. Neubuerger's Foreword.

J. E. ASH
Colonel, Medical Corps
Curator

TABLE OF CONTENTS

(Accession number is 78842 unless otherwise stated)

GANGLION CELL CHANGES IN A SENILE BRAIN

Nissl Stain

CLINICAL NOTE: A mentally confused 75-year-old male who fell off the roof of his hotel. He was found unconscious and in shock. Death occurred within a few hours. (A 3544)

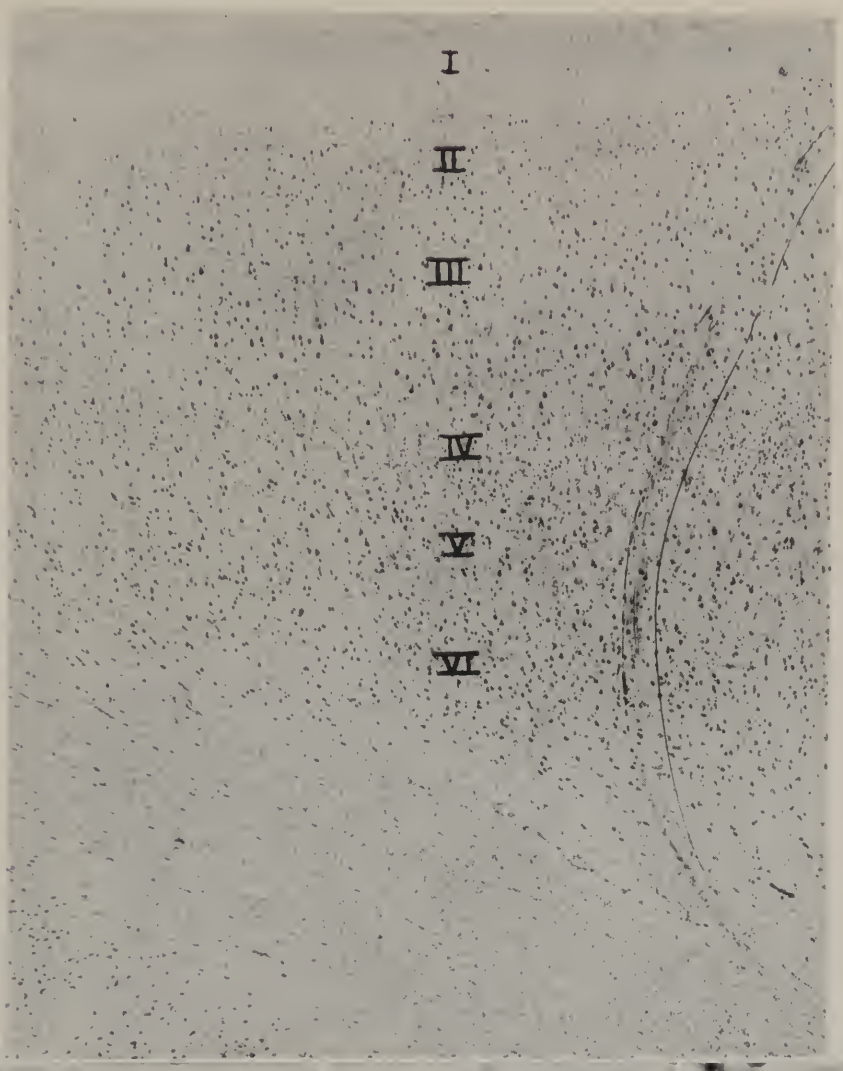
PATHOLOGY: There were multiple bone fractures. The cerebral cortex was somewhat narrowed and the ventricles were dilated. Numerous fat emboli were noted. The cerebral blood vessels were not appreciably sclerotic.

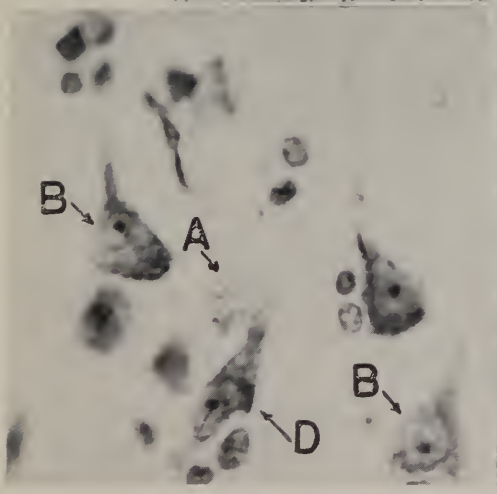
The section shows a diffuse reduction in the number of neurons in the cortex, particularly in the IIId layer. Many of the ganglion cells in this layer are altered. Some are in the late stage of dissolution (A). Others have cytoplasm that is pale and irregularly granular. Many of the nuclei are eccentric (B). Here and there some of the cells are shrunken, have dark-stained cytoplasm, and homogeneous nuclei with eccentric nucleoli.

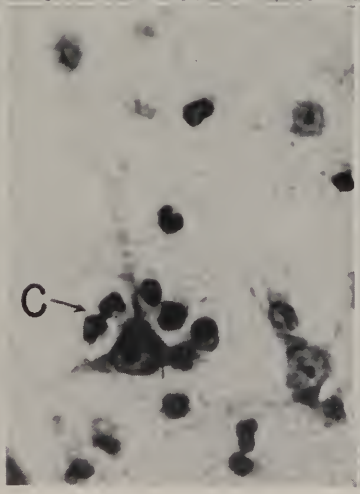
Layers V and VI show numerous round cells (mostly oligodendroglia) around ganglion cells: some constitute satellitosis (C), others neuronophagia (D). (See also Slide 2 from the same case.)

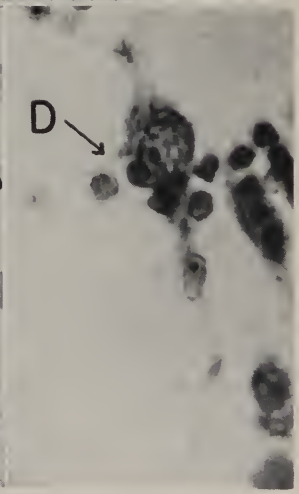
Reference: Weil, A.: A text-book of neuropathology, Philadelphia, Lea & Febiger, 1933, pp. 30-38.

SLIDE 1. GANGLION CELL CHANGES IN A SENILE BRAIN









NEG. 73753 X 40 NEG. 73796 X 680 NEG. 73910 X 680 NEG. 73909 X 680

ARGENTOPHIL PLAQUES IN A SENILE BRAIN

von Braunmuehl Silver Stain

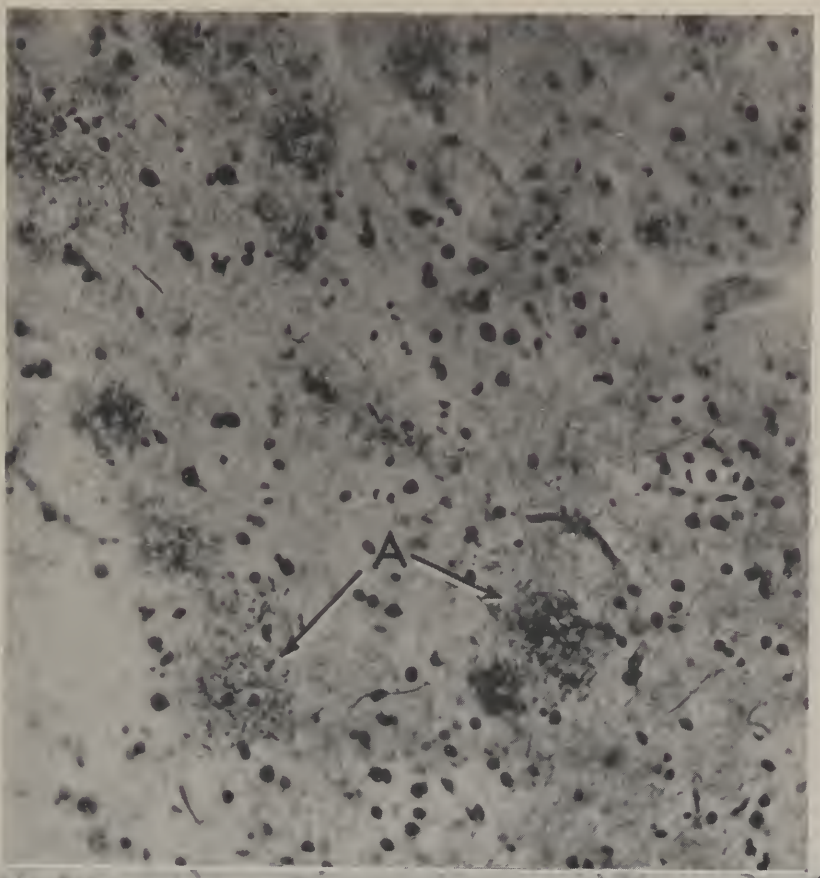
CLINICAL NOTE: Same case as Slide 1. (A 3544)

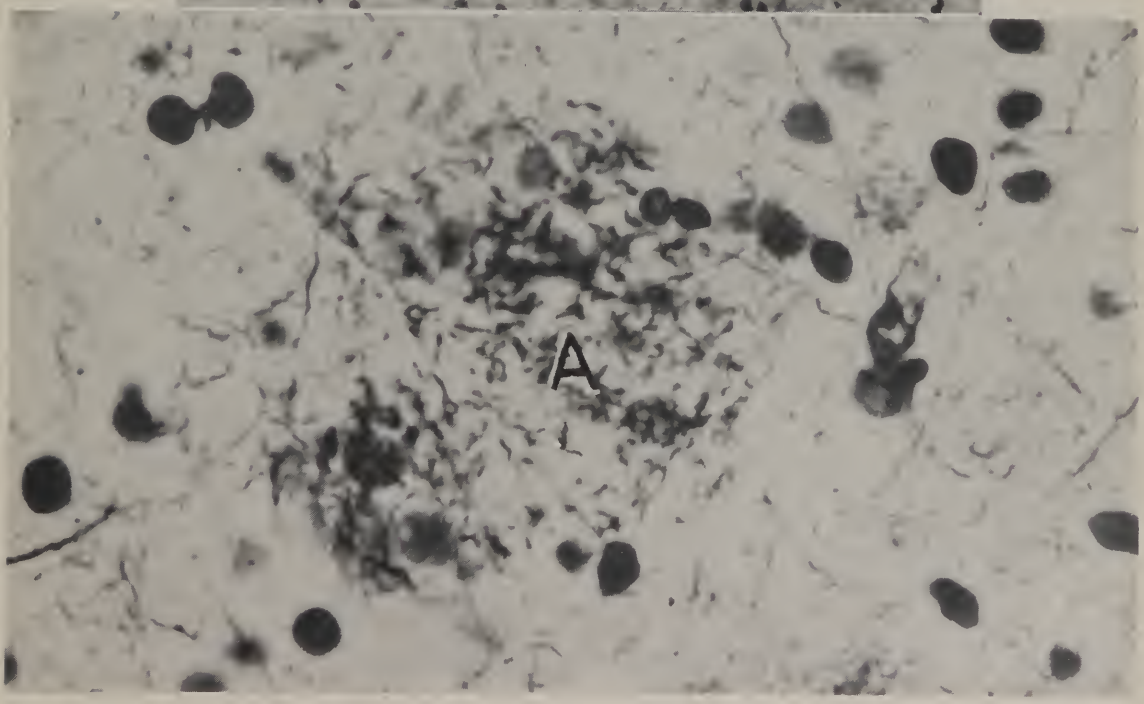
PATHOLOGY: A section taken from the frontal cortex shows numerous typical argentophil "senile plaques" (A). The reticulum of the plaques is denser and of larger caliber than that of the glial cells. The central portion of most of the plaques is clear but in some of the smaller plaques the central region contains a shrunken nucleus-like mass. As is virtually always the case, the plaques are unrelated to blood vessels.

It is thought that senile plaques may represent a local metaplasia of glial reticulum.

Reference: Hassin, G. B.: Histopathology of the peripheral and central nervous systems, ed. 2, New York, Hoeber, 1940, pp. 374-377.

SLIDE 2. ARGENTOPHIL PLAQUES IN A SENILE BRAIN





NEG. 72765a X 250 NEG. 72765b X 1000

ALZHEIMER'S DISEASE

von Braunmuehl Silver Stain

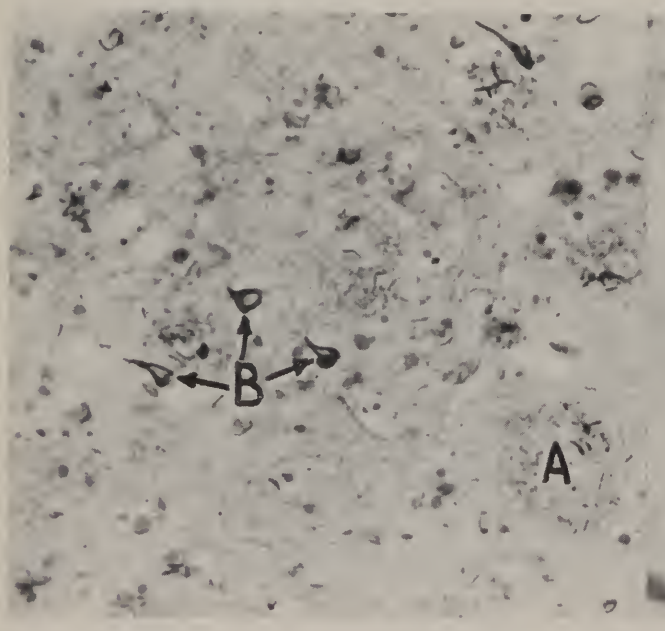
CLINICAL NOTE: Not available. (NP 141)

PATHOLOGY: The cortex is atrophied and strewn with senile plaques (A). In some of the ganglion cells the cytoplasm and processes contain argentophil filamentous elements (of Alzheimer) (B). The filaments are relatively thick and take the form of intricate twists and spirals. Simpler patterns are also noted.

Since the filamentous elements occur independently of the preformed neurofibrillar frame it has been concluded that they are not derived from neurofibrils.

These filaments are seen in senile involution psychoses including presenile dementia.

Reference; Bielschowsky, M.: Histopathology of nerve cells. In Penfield, W., Cytology and cellular pathology of the nervous system, New York, Hoeber, 1932, vol. 1, pp. 179 & 189. 179 & 180.



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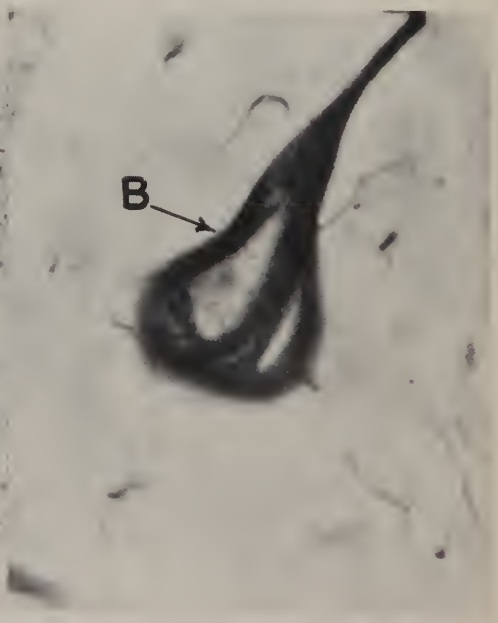


NEG. 76859 X 1890

PICK'S DISEASE

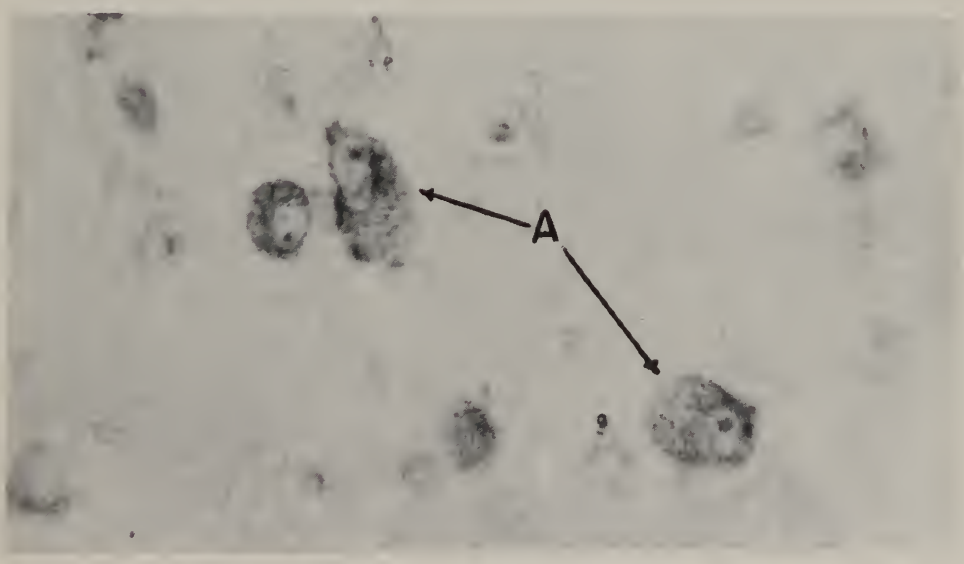
Nissl Stain

CLINICAL NOTE: Not available. (NP 153)

PATHOLOGY: In the upper layers of the cortex many ganglion cells have been replaced by astrocytes. The remaining ganglion cells show varying degrees of change. Many contain cytoplasmic granules that balloon the cell and push the nucleus over against the cell wall (A). Other ganglion cells are shrunken, have "moth-eaten" cytoplasm and tortuous apical dendrites. The white matter contains many more cells than normally, most of these being astrocytes and oligodendroglia.

The slide lacks the senile plaques seen in the case of Alzheimer's disease illustrated in Slide 3; such structures are, however, occasionally present in Pick's disease.

Reference: Alexander, L., and Looney, J. M.: Histologic changes in senile dementia and related conditions, Arch. Neurol. & Psychiat. 40: 1075, 1938.



NEG. 72713 X 1000

PERTUSSIS ENCEPHALOPATHY

Nissl Stain

CLINICAL NOTE: A boy 16 months of age who had had whooping cough for five weeks prior to hospitalization. On admission the paroxysms of cough were associated with periods of unusually severe apnea and cyanosis. Fever was high. During the three days before death there were repeated muscular twitchings and generalized convulsive seizures. Death occurred during a convulsion. (A 3707)

PATHOLOGY: Autopsy showed a patchy bronchopneumonia and chronic suppurative otitis media. Grossly the brain appeared to be normal.

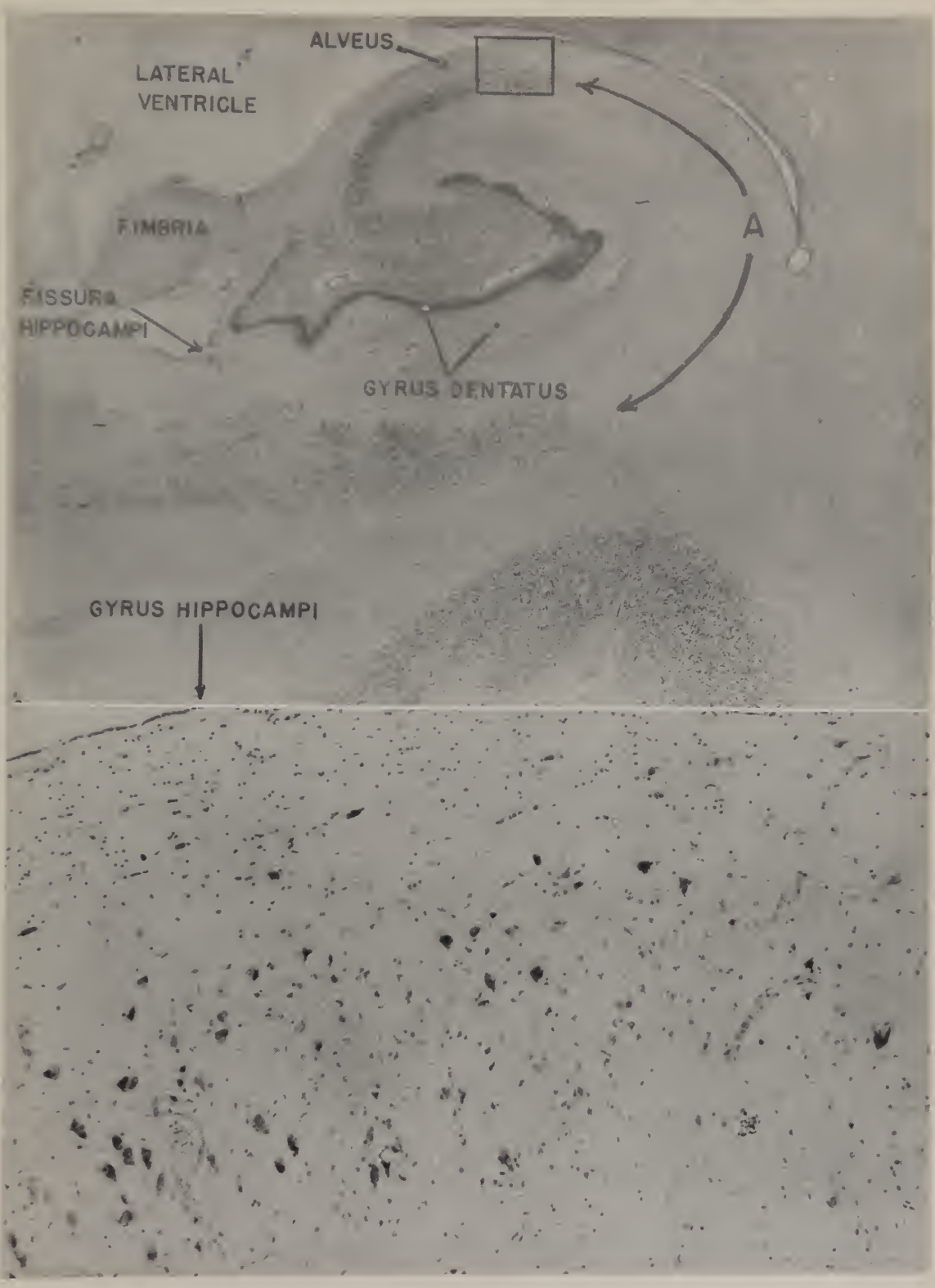
The section shows virtually complete disappearance of a part of the pyramidal cell layer of the hippocampus known as Sommer's sector (A). The changes in the remaining cells of this layer vary from slight vacuolization of the cytoplasm to ghost-like transformation of the entire cell. These features are especially evident in the zone of transition between degenerated and relatively normal pyramidal layer (see lower photograph). Many of the nuclei are in a state of dissolution (karyolysis) others show hyperchromatosis and rupture of the nucleus (karyorrhexis). No glial reaction is observed. The gyrus dentatus is virtually intact.

Extensive degenerative changes are visible also in the temporal cortex lamination being scarcely discerned. Shrunken pale ganglion cells are common. No inflammatory cells are noted. In some areas the vessel walls are hyperplastic.

This picture is also referred to as "toxic degenerative disease of the brain". Wilson speaks of the condition as "pertussis eclampsia".

Reference: Hiller, F.: Die Zirkulationsstörungen des Rückenmarks und Gehirns. In Bumke, O., and Foerster, O., Handbuch der Neurologie, Berlin, Springer, 1936, vol. 11, pp. 277-289.

SLIDE 5. PERTUSSIS ENCEPHALOPATHY



NEG. 72722 X 10 NEG. 76853 X 150

SPONGIOBLASTOMA POLARE

Hematoxylin and Eosin Stain

CLINICAL NOTE: A 45-year-old male who presented the clinical picture of bulbar paralysis. (A 3693)

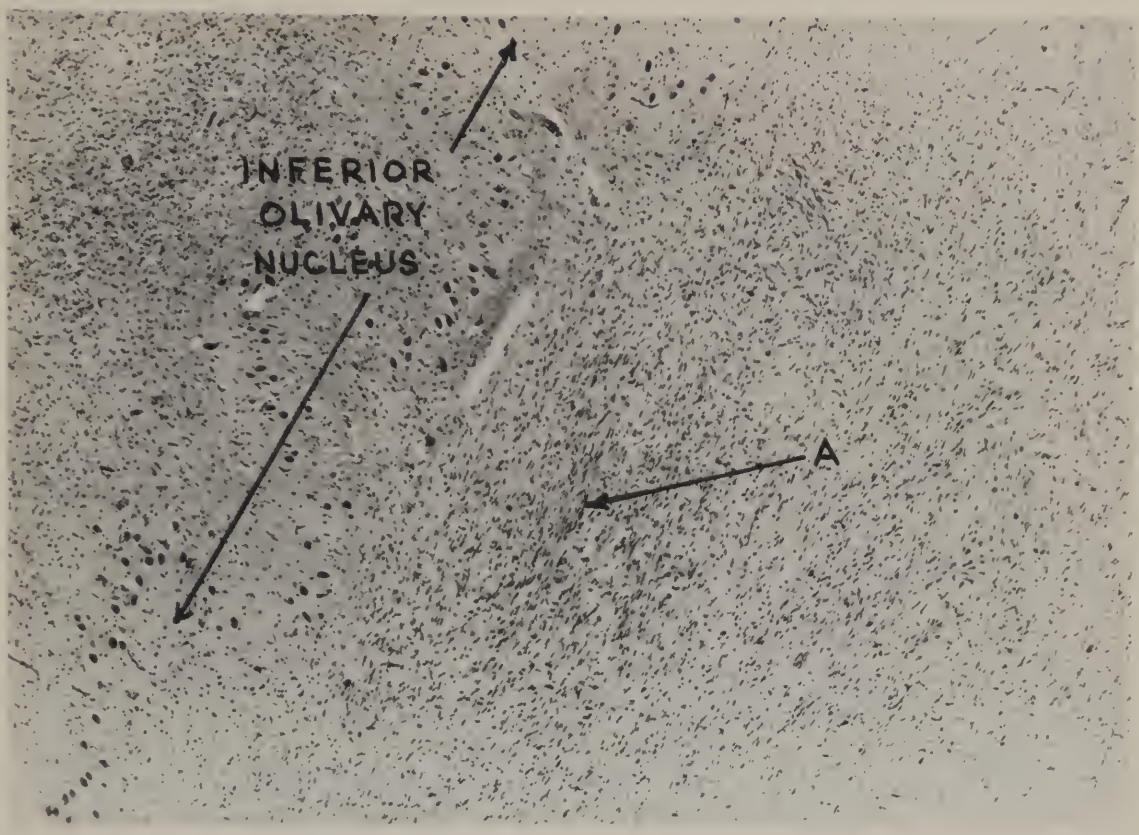
PATHOLOGY: The upper portion of the medulla oblongata was diffusely enlarged to about three times the normal size. Section of the gross specimen disclosed a diffuse tumor which had eradicated all landmarks except the inferior olivary nucleus (see photograph).

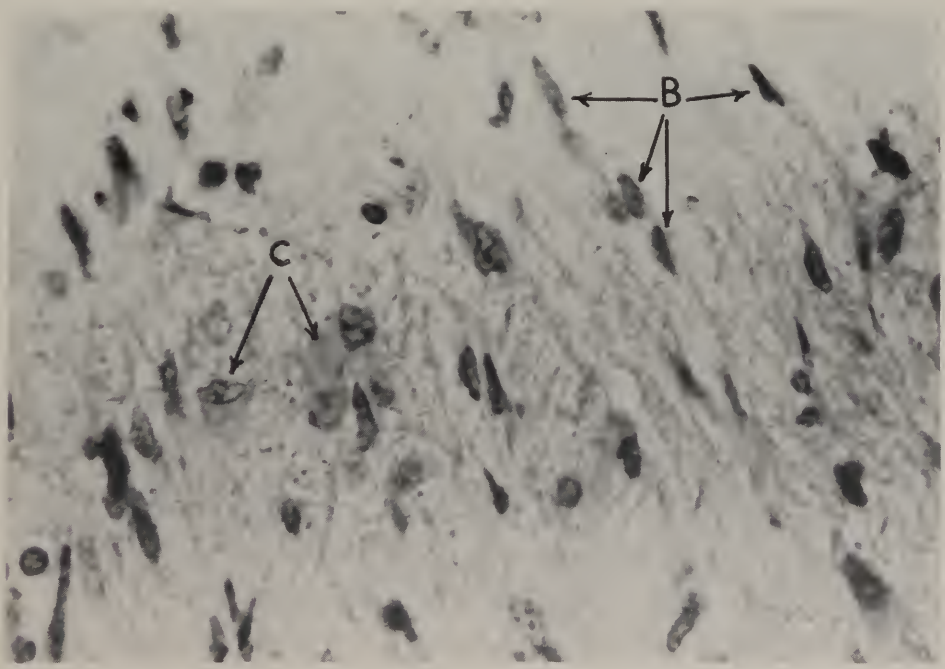
Histologically the tumor is without discernible boundaries. It is composed of elongated cells frequently assembled in fasciculi (A). The nuclei are mostly elongated or round, depending on the plane of section. The cytoplasm of the cells is scanty and trails off into wavy processes which are either unipolar or bipolar (B). There are no areas of degeneration, and blood vessels are scant. Mitoses were not encountered. Here and there one notes larger cells with vesicular nuclei and homogeneous cytoplasm; these are mostly plump astrocytes (C).

This type of tumor has a predilection for the brain stem, particularly the walls of the IIId and IVth ventricles.

Reference: Pilcher, C.: Spongioblastoma polare of the pons, Arch. Neurol. & Psychiat. 32: 1210, 1934.

SLIDE 6. SPONGIOBLASTOMA POLARE





NEG. 72716 X 50 NEG. 73886 X 680

GENERAL PARESIS

(Rod Cells)

Hortega Stain

CLINICAL NOTE: Not available.

PATHOLOGY: The section is taken from the hippocampal formation and adjoining temporal cortex. Throughout the section there are many elongated microglia (A). Their nuclei are long and straight or curved. In most of the cells the perinuclear cytoplasm is scanty and is continued into rather broad polar processes with sparse collaterals. Microglia elsewhere have richly arborizing processes.

Reference: Hortega, P. del Rio: Microglia. In Penfield, W., Cytology and cellular pathology of the central nervous system, vol. 2, New York, Hoeber, 1932, pp. 483-534.

NEG. 76611 X 435

Slide 7

ENCEPHALOMALACIA (EARLY STAGE)

Nissl Stain

CLINICAL NOTE: A 24-year-old male who had been beaten on the head and robbed nine months before death. He had a marked depression of the skull in the right parietal region. Gradually he became irritable and moody. Five weeks prior to death he had an attack of projectile comiting. Periods of unconsciousness supervened. The spinal fluid was bloody and under increased pressure. Toward the end he had muscular twitching and convulsions. (A 3704)

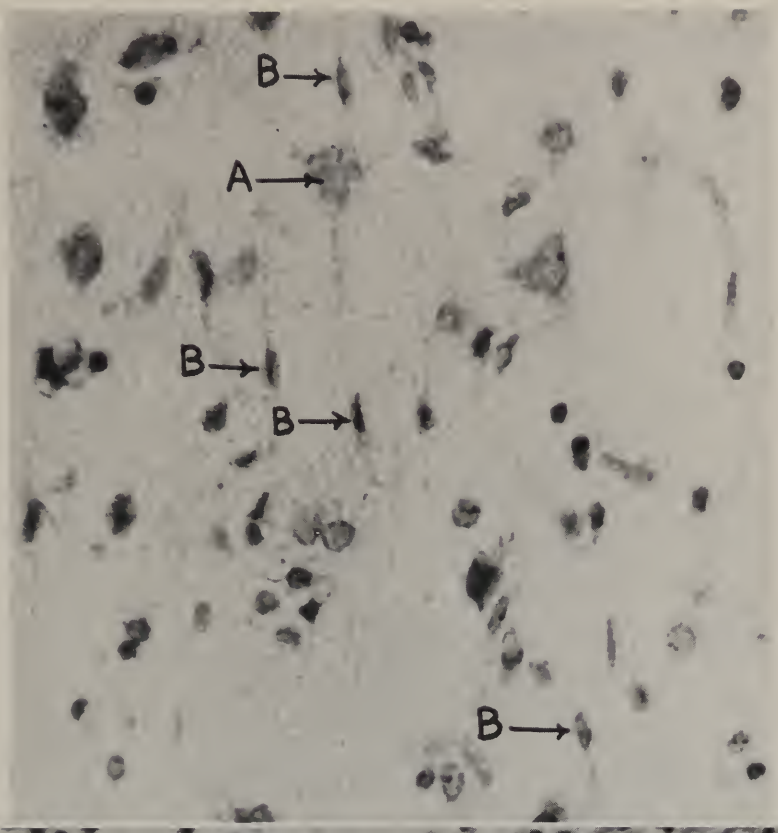
PATHOLOGY: The immediate cause of death was a massive bilateral pulmonary embolism. Widespread subarachnoid hemorrhage was present. In the right parietal lobe there was a sunken scarred area; the left parietal lobe contained a focus of ischemic softening. Petechiae and red infarctions were noted in both frontal lobes and in both gyri cinguli.

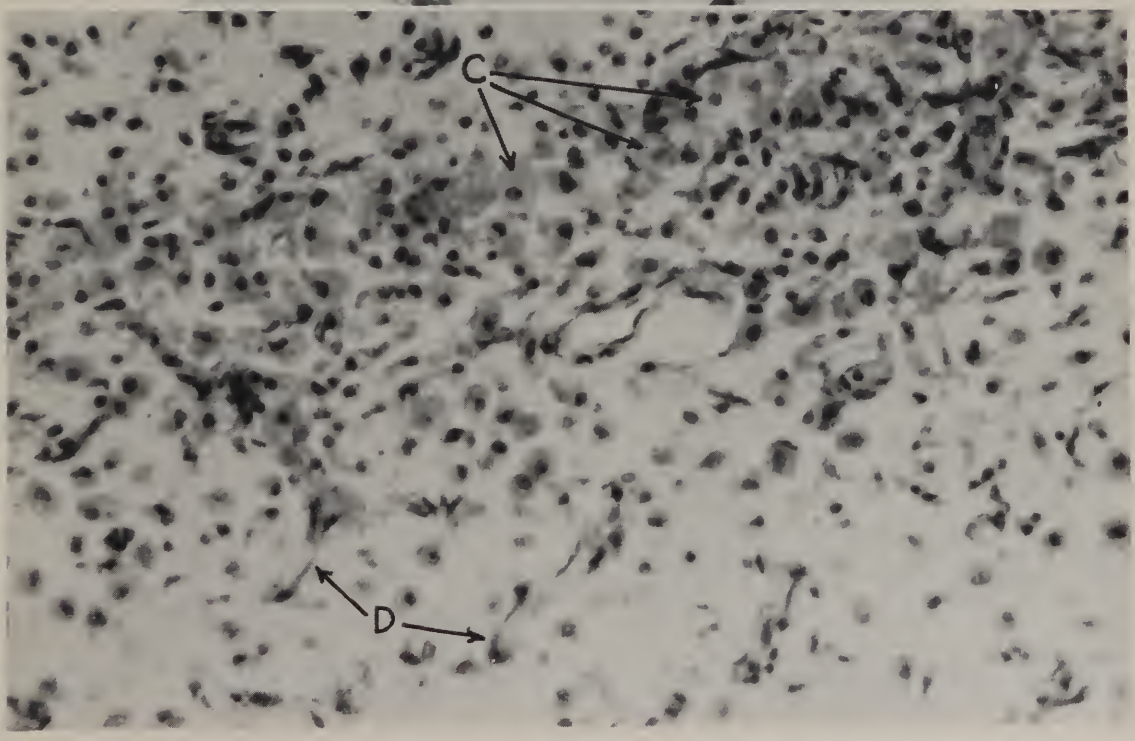
The slide shows a variety of changes. In the cortex many of the ganglion cells have disappeared and numerous others have become ghost-like (A). Numerous rod cells (Stäbchenzellen) are visible (B). Swollen astrocytes and gitter cells (compound granular corpuscles) (C) are also noted. In various areas there is extensive proliferation of capillaries (D).

Other sections from this case are shown in Slides 53 and 60.

Reference: Rand, C. W., and Courville, C. B.: Histologic studies of the brain in cases of fatal injury to the head, Arch. Neurol. & Psychiat. 36: 1277, 1936.

SLIDE 8. ENCEPHALOMALACIA (EARLY STAGE)





NEG. 72751a X 600 NEG. 72751b X 350

CEREBRAL AND MENINGEAL CHANGES FOLLOWING SAGITTAL SINUS THROMBOSIS

Hematoxylin and Eosin Stain

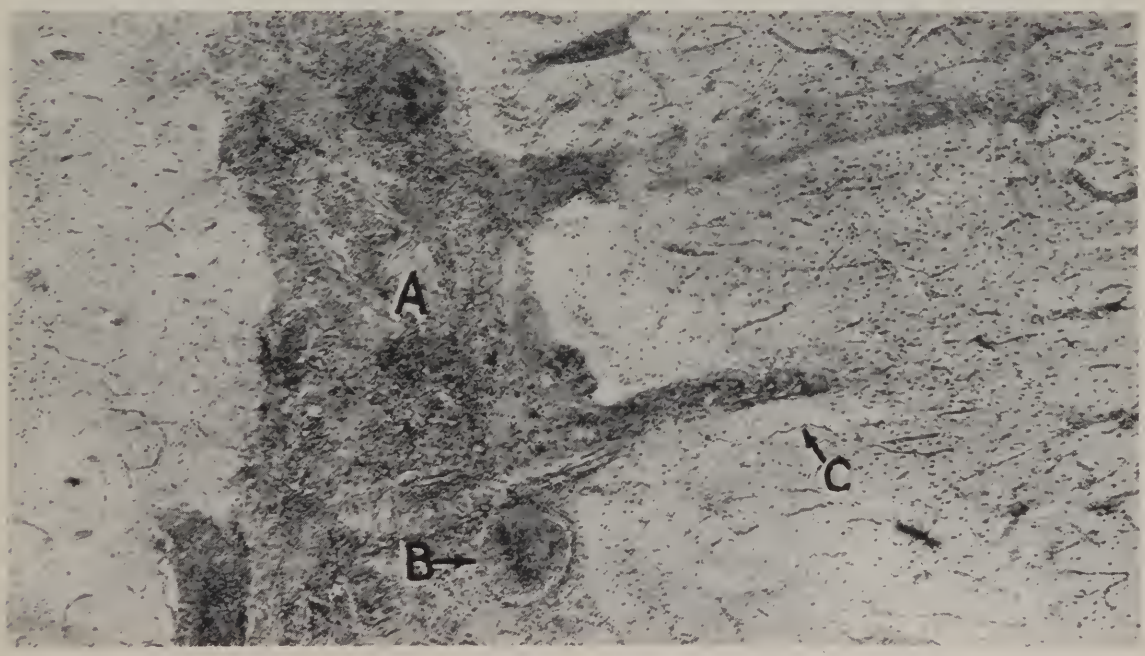
CLINICAL NOTE: A 23-year-old woman with puerperal infection, who became mentally confused and who developed high fever and had repeated convulsions. (A 3361)

PATHOLOGY: There was thrombosis of the sagittal sinus and of numerous veins in the leptomeninges. The adjacent cerebrum was the seat of multiple red softened areas.

The leptomeninges are greatly edematous and hemorrhagic (A). A number of the vessels are recently thrombosed (B). Vessels penetrating the cortex are greatly distended, as is also the cortical capillary bed. Hemorrhages are small and numerous. Around the larger vessels in the cortex there are diffuse spongy pale-staining patches indicative of edema (C). The ganglion cells are in a state of early necrosis; many of them stain pink instead of having the blue tint seen normally. The small bluish globules, most of which are in the white matter, are artefacts.

See also Slide 22 from same case.

Reference: Doyle, J. B.: Obstruction of the longitudinal sinus, Arch. Neurol. & Psychiat. 18: 374, 1927.



Slide 9 NEG. 72719 X 50

CELLULAR RESPONSE SEVERAL WEEKS AFTER CEREBRAL HEMORRHAGE

Hematoxylin and Eosin Stain

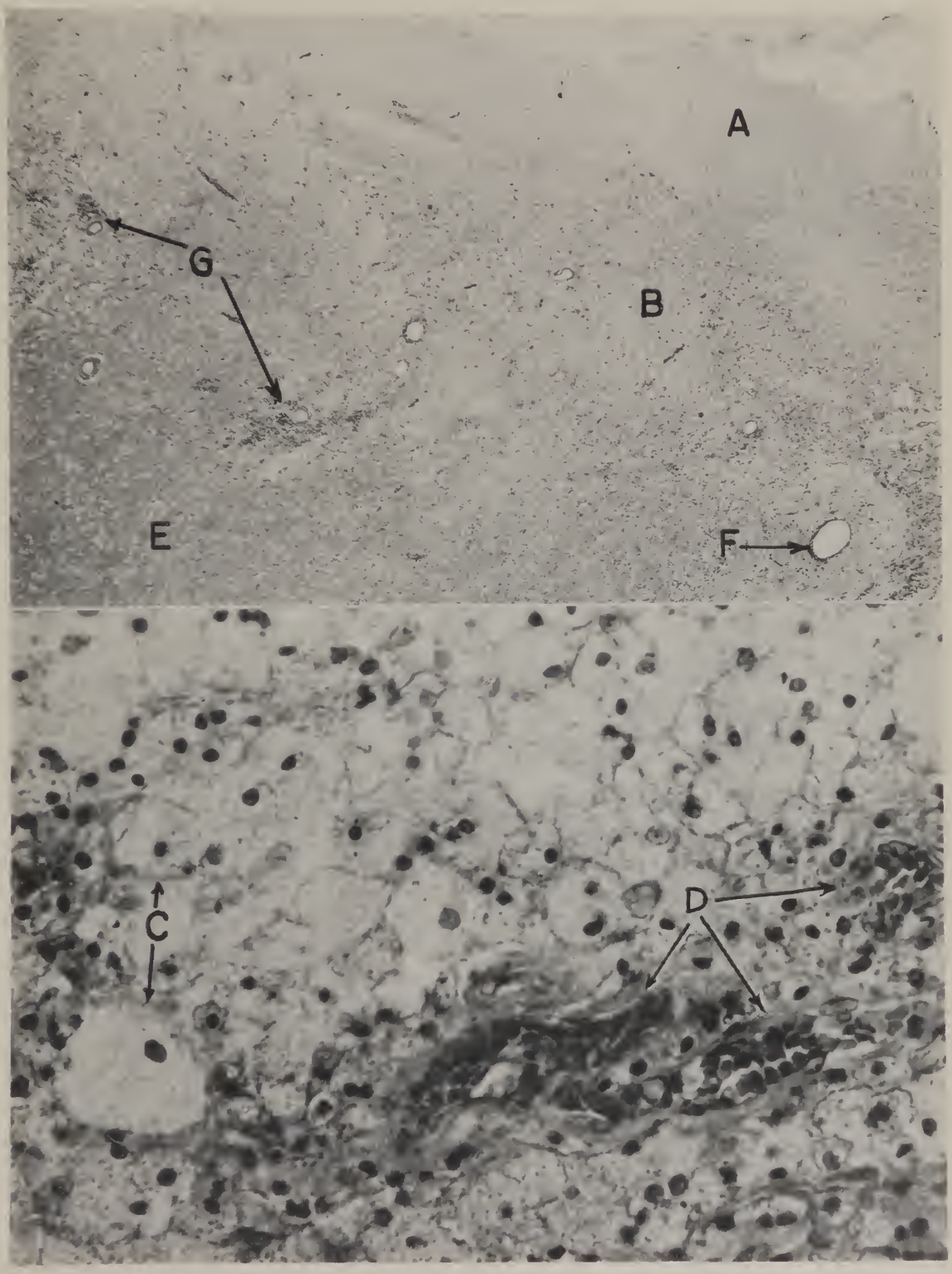
CLINICAL NOTE: A 57-year-old woman had suffered from severe hypertension for years. She had a "cerebral accident" manifested by hemiplegia to which she succumbed after several weeks. (A 2934)

PATHOLOGY: There was a large blood clot occupying the left internal capsule and basal ganglia.

The slide shows necrotic tissue (A) at the edge of the clot. Adjacent to the necrotic tissue is a thick reactive zone (B), composed essentially of gitter cells (C) and proliferated blood vessels (D). The border between the reactive layer and parenchyma is fairly sharp. The parenchyma in the vicinity of the reactive layer has a moth-eaten appearance which is to be ascribed chiefly to edema (E). The parenchymal blood vessels are surrounded by round cells - in some instances as cuffs (F) and in others as asymmetrical collections surrounded in turn by plump astrocytes (G). Throughout the section the arterioles show moderate sclerosis.

Reference: Oppenheimer, B. S., and Fishberg, A. M.: Hypertensive encephalopathy, Arch. Int. Med. 41: 264, 1928.

SLIDE 11. CELLULAR RESPONSE SEVERAL WEEKS AFTER CEREBRAL HEMORRHAGE



NEG. 74152 X 100 NEG. 74150 X 600

ARTERIOLAR SCLEROSIS WITH PERIVASCULAR HEMORRHAGES

Hematoxylin and Eosin Stain

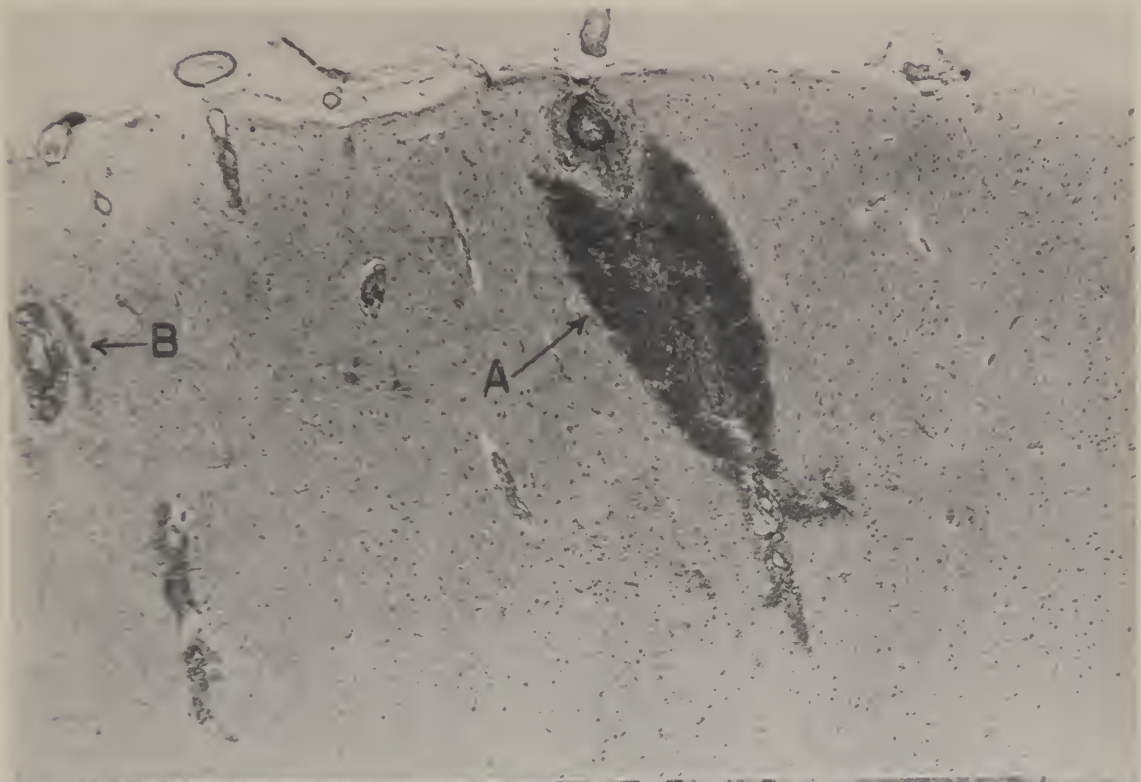
CLINICAL NOTE: A 55-year-old woman who was subject for many years to high blood pressure. Frequently the pressure was found to be subnormal. One day she fell unconscious, went into coma, and remained so several days before death. (A 3312)

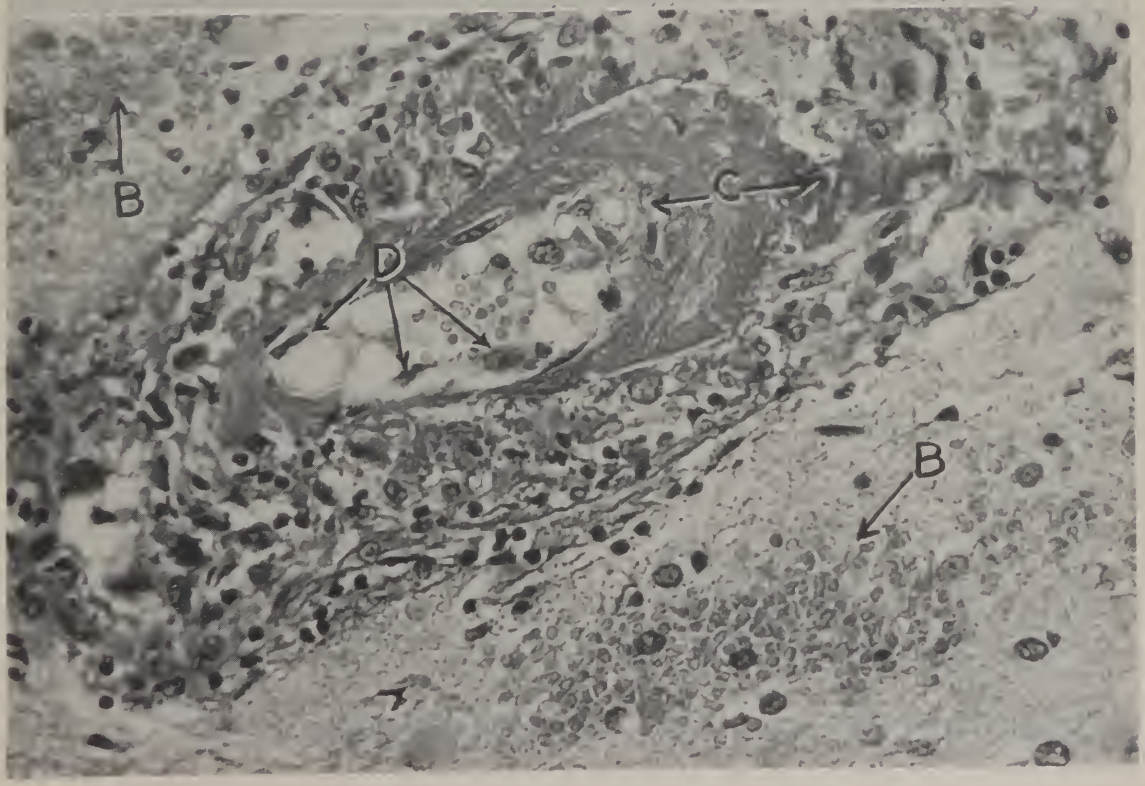
PATHOLOGY: Grossly there were many petechial hemorrhages in the leptomeninges and in the cortex, especially of the frontal lobes.

Virtually all the cerebral hemorrhages are perivascular (A). Some are ring hemorrhages (B). Subpial hemorrhages are also present. The walls of the arterioles have undergone marked hyaline change (C). Proliferation of intimal cells is also noted (D). The adventitia is loculated and infiltrated with round cells. A lesser number of round cells are to be found in the perivascular spaces. The arteriolar changes are also evident in the meninges. Cortical ganglion cells show a mild degree of ischemic change.

Reference: Hassin, G. B.: Histopathology of the central and peripheral nervous systems, New York, Hoeber, 1940, pp. 330-334.

SLIDE 12. ARTERIOLAR SCLEROSIS WITH PERIVASCULAR HEMORRHAGES





NEG. 72764a X 30 NEG. 72764b X 435

CEREBRAL LACUNAE (ETAT LACUNAIRE DU CERVEAU)

Hematoxylin and Eosin Stain

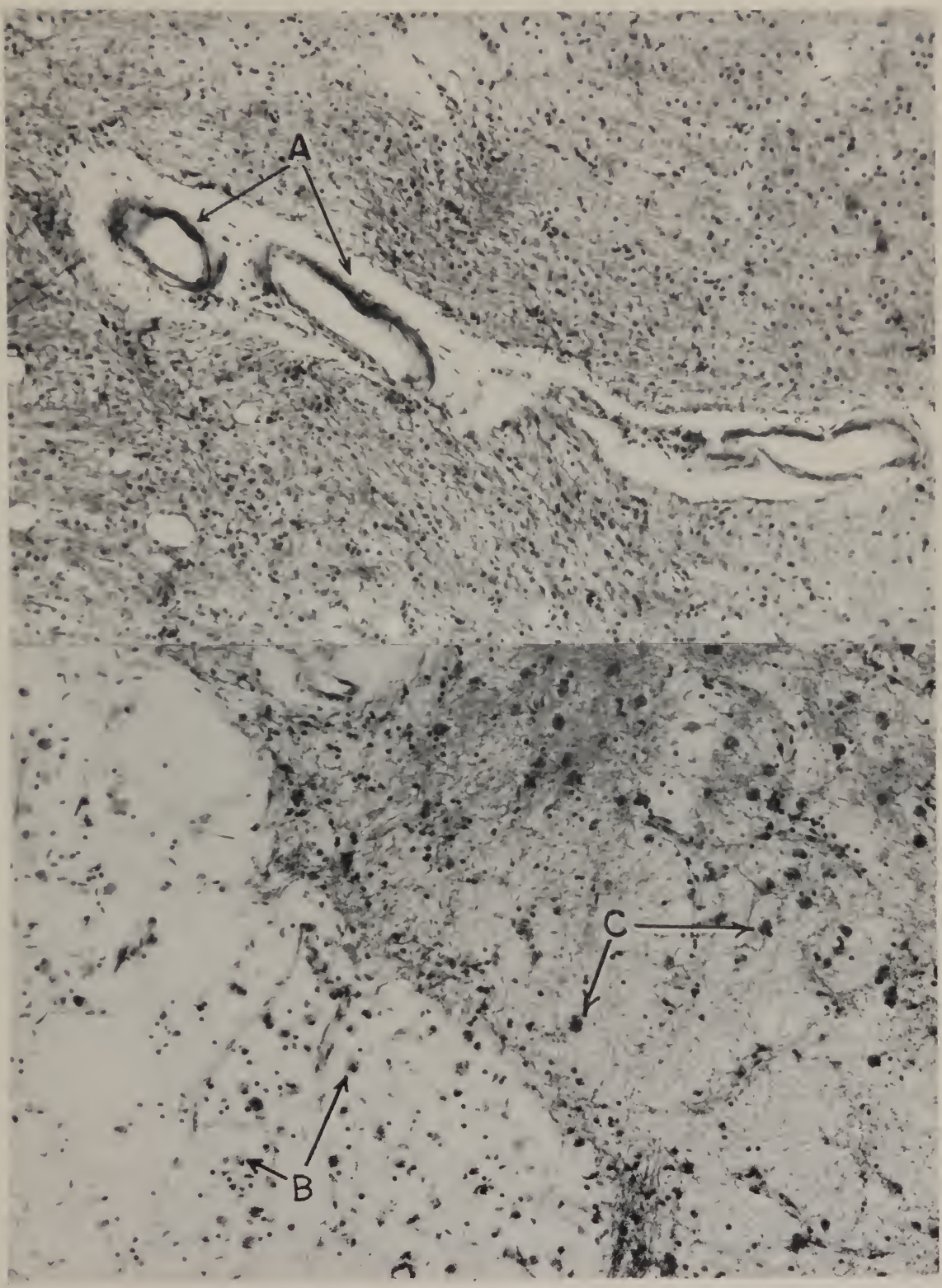
CLINICAL NOTE: A 52-year-old woman had suffered for about a year from symptoms characteristic of the "syndrome lacunaire" (of Pierre Marie): dysarthria, spasticity, ataxic gait, movement forward with very short steps, periodic choreatic and athetotic movements, and disturbances of sensibility. The patient was also blind and disoriented. (A 3371)

PATHOLOGY: Necropsy revealed evidence of marked hypertensive cardiovascular disease. The basal ganglia, thalamus and subcortical white matter contained many old and recent small softenings, tissue gaps and hemorrhages.

The arterioles are sclerotic but their lumina are patent (A). It has been suggested that the focal destruction of the brain substance is secondary to adventitial changes. Larger tissue gaps are permeated by a fine trabecular network. Within the meshes of such gaps there are moderate numbers of gitter cells (B). The surrounding walls are composed of proliferated astrocytes (C) as well as round cells and pigment-laden macrophages.

Reference: Grasset, J.: La cerebrosclerose lacunaire progressive d'origine arterielle, Semaine med. 24: 329, 1904.

SLIDE 13. CEREBRAL LACUNAE (ETAT LACUNAIRE DU CERVEAU)



NEG. 72764 X 435 NEG. 72747 X 150

RECENT CORTICAL NECROSIS

Nissl Stain

CLINICAL NOTE: Not available.

PATHOLOGY: Microscopically, the cortex shows areas of "paleness" of varying degree. In one part of the section there is such a marked dropping out of cells that lamination can no longer be detected. In other areas the necrosis is focal (A).

In the areas of the more widespread necrosis many of the ganglion cells are no longer recognizable while others can be identified by their pale triangular remnants of nuclei. Reactive gliosis is sparse, and the development of granulation tissue insignificant.

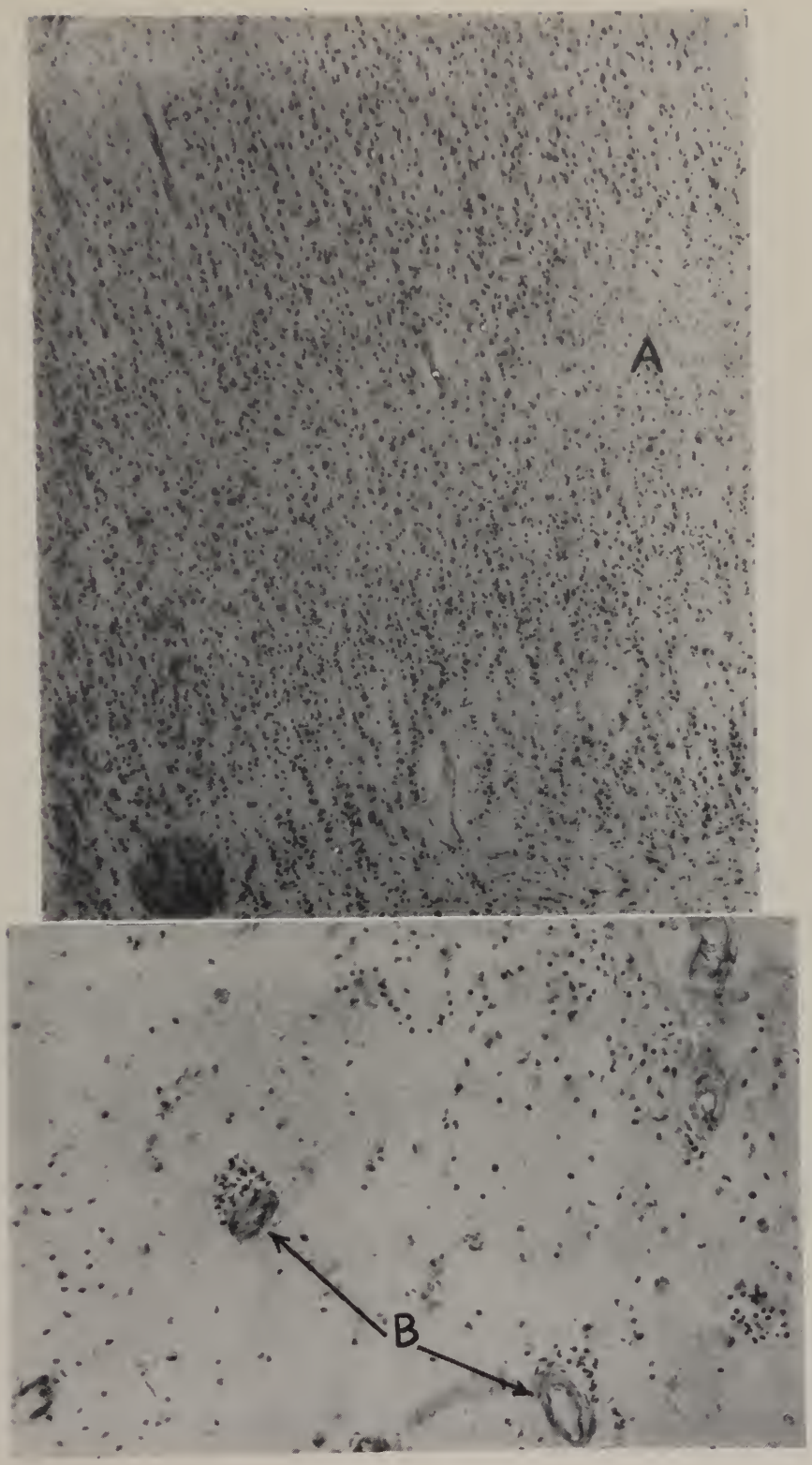
As is frequently seen in the earlier stages of ischemic necrosis, the capillaries in the ischemic areas are surrounded by polymorphonuclear leukocytes; this is particularly striking in the uppermost layer of the cortex (B).

In the more involved regions the pia-arachnoid contains polymorphonuclear leukocytes; in less involved parts the predominant meningeal cell is the lymphocyte.

The ischemic changes are present also in the white matter.

Reference: Weil, A.: A text-book of neuropathology, Philadelphia, Lea & Febiger, 1933, pp. 80-87.

SLIDE 14. RECENT CORTICAL NECROSIS



NEG. 72721 X 70 NEG. 73028 X 200

EARLY ISCHEMIC CHANGES IN THE HIPPOCAMPUS IN A CASE OF APOPLEXY

Nissl Stain

CLINICAL NOTE: From same case as Slide 10. (A 3460)

PATHOLOGY: This section is taken from the hippocampus and adjoining temporal gyri. The changes in Sommer's sector of the pyramidal layer of the hippocampus are striking. Many of its cells show early ischemic changes: paleness (A), elongation of the cell body and other distortions (B), tortuosity of dendrites (C), triangulation and pyknosis of nuclei, neuronophagia (D), and so on. There are focal perivascular collections of polymorphonuclear leukocytes in ischemic areas but they are present to considerably less degree than in Slide 14.

The adjoining temporal gyri are similarly affected but to a lesser degree.

Similar and even more pronounced changes in the hippocampus may be found in such conditions as eclampsia of pregnancy (Slide 88), yellow atrophy of the liver (Slide 77), schizophrenia (Slide 43), epilepsy (Slide 41), focal embolic encephalitis (Slide 28), general paresis (Slide 26), and whooping cough (Slide 5).

Reference: Hiller F.: Die Zirkulationsstörungen des Rückenmarks und Gehirns. In Bumke, O., and Foerster, O., Handbuch der Neurologie, vol. 11, pp. 278-280, 1936.

SLIDE 15. EARLY ISCHEMIC CHANGES IN THE HIPPOCAMPUS IN A CASE OF APOPLEXY

NEG. 72752 X 355 NEG. 73973 X 440

ISCHEMIC NECROSIS OF BRAIN

Hematoxylin and Eosin Stain

CLINICAL NOTE: A male 55 years old who was admitted to hospital confused and unresponsive. Coma became profound. There was a hemiplegia on the left side. Death occurred three days after admission. (A 3073)

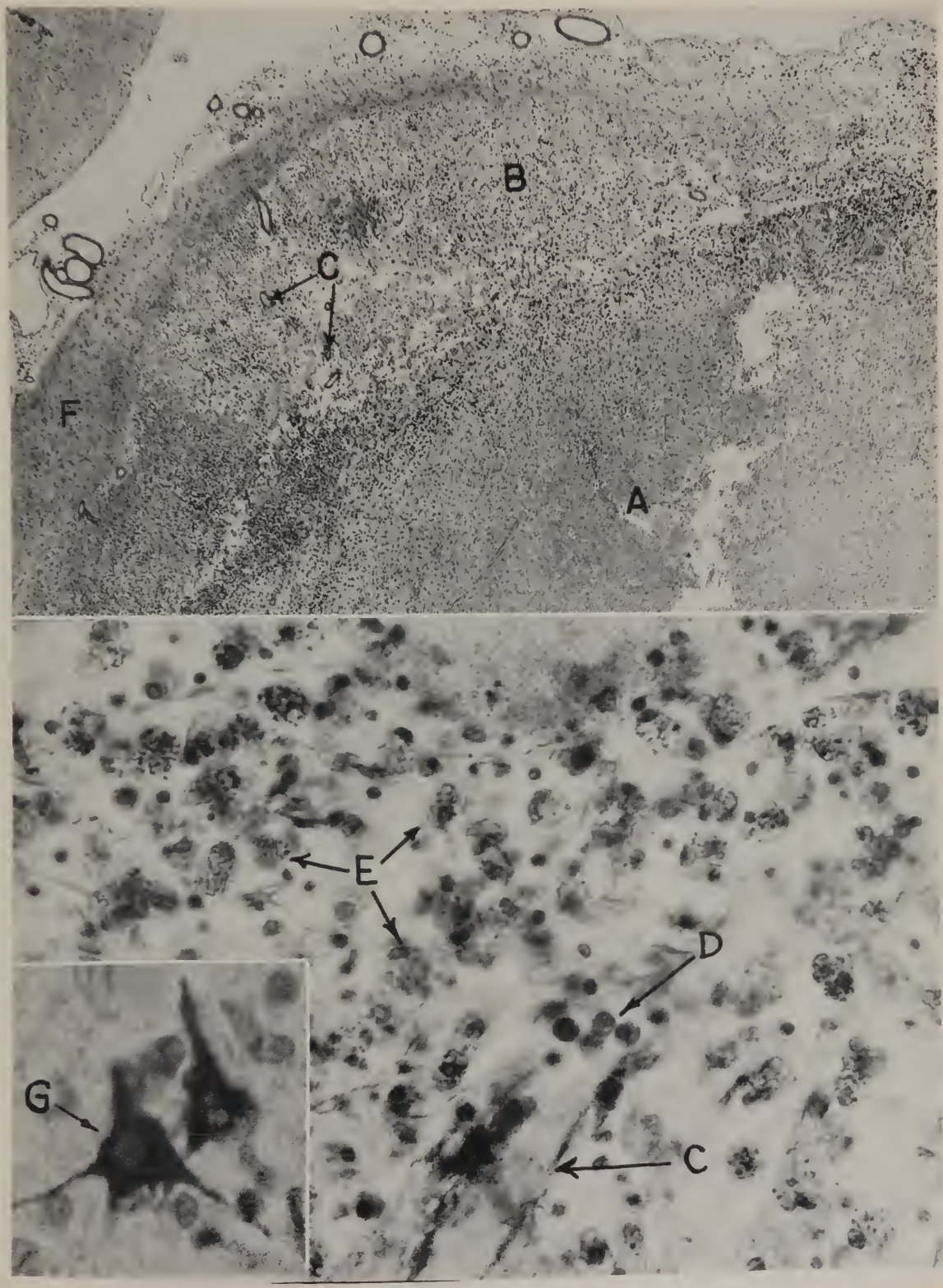
PATHOLOGY: Autopsy revealed a generalized severe arteriosclerosis. The right parietal lobe contained a softened area.

In the section one notes a subcortical excavation, the deeper portion of which is completely necrotic (A). In the superficial parts of the softened area (B) the reactive process is in full swing. New vessels have appeared (C). Numerous small round cells (D), many pigment-containing gitter cells (E), and fibroblasts are scattered through this area. A few giant cells are noted. Proliferated plump astrocytes (F) are most numerous in the uppermost layer of the cortex.

An incidental finding is the presence of calcified nerve cells (G) in an adjacent gyrus.

Reference: Stern, K.: The pathology of apoplexy, J. Neurol. & Psychiat. 1: 26, 1938.

SLIDE 16. ISCHEMIC NECROSIS OF BRAIN



NEG. 73979 X 30 NEG. 72762D X 550 NEG. 72762a X 500

OLD CEREBRAL SOFTENINGS WITH CYSTIC TRANSFORMATION

Nissl Stain

CLINICAL NOTE: The patient, a male aged 62 years, had had a severe trauma, the details of which are not available. Three years later he died. (A 3505)

PATHOLOGY: At autopsy an old thrombotic occlusion of the right middle cerebral artery was found. Associated with it were old softened areas in the parietal lobe and basal ganglia. A similar condition was found in the cerebellum.

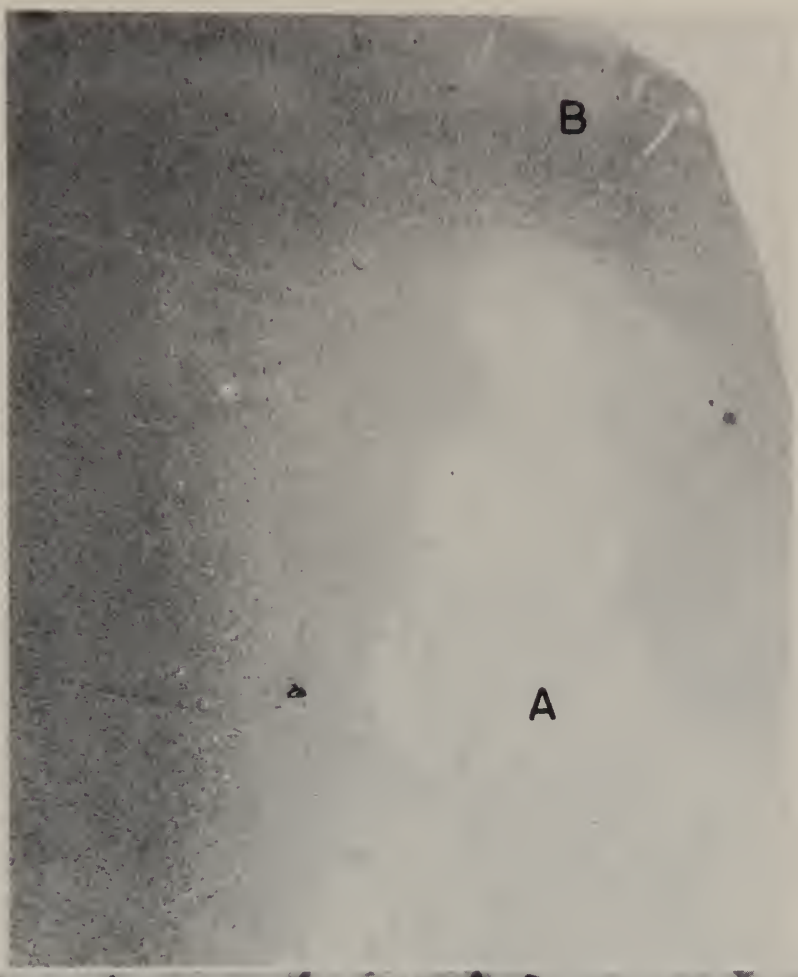
An extensive softened area is present in the white matter (A). The cortex (B) is relatively intact. Strands of tissue, varying in thickness, bridge the gap, forming a number of cyst-like spaces which communicate freely with one another. The meshes of the softened area contain many gitter cells (C). The wall surrounding the focus is permeated with plump astrocytes (D). There is virtually no proliferation of vessels.

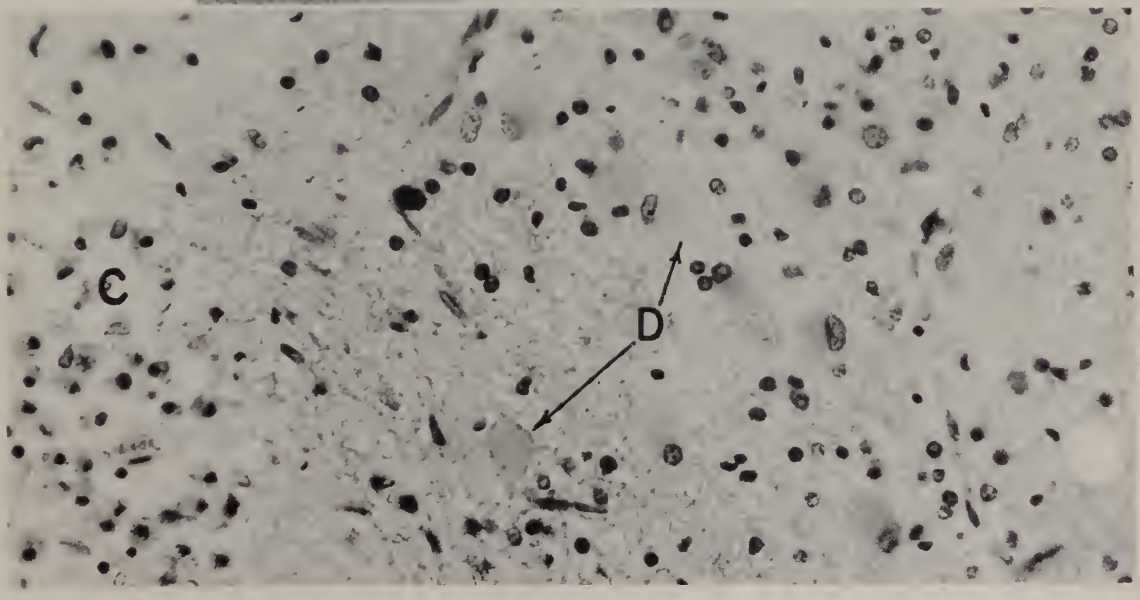
In contrast to the etat lacunaire shown in Slide 13, there are no vessels with free lumina spanning the tissue gaps.

Note: Slide 17 of some of the sets is from the cerebellum. In the latter structure the lesion is of about the same age as that in the cerebrum. The changes in the cerebellum are present mostly in the white matter but entire folia are also involved.

Reference: Singer, H. D.: Histopathology of focal brain softening, Am. J. Psychiat. 3: 717, 1924.

SLIDE 17. OLD CEREBRAL SOFTENINGS WITH CYSTIC TRANSFORMATION





NEG. 73978 X 20 NEG. 72763 X 350

VERRUCOUS ATROPHY OF THE CEREBRAL CORTEX

Nissl Stain

CLINICAL NOTE: The patient, a 49-year-old psychotic woman, who for several years had had symptoms of a heart ailment of undisclosed nature. (A 3538)

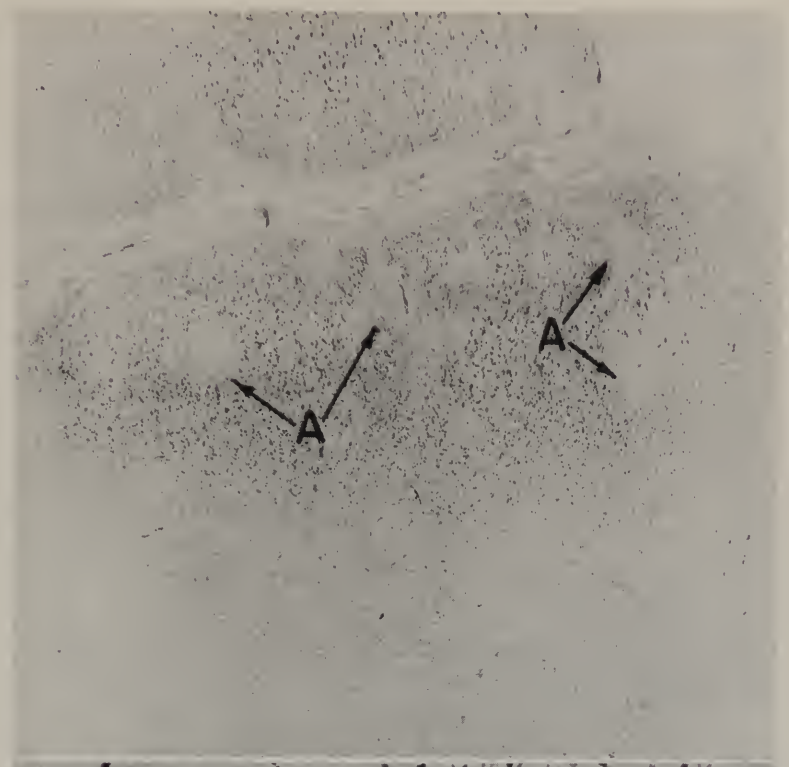
PATHOLOGY: Autopsy revealed an endocarditis affecting the mitral and aortic valves, regarded as rheumatic in origin. In the parietal lobe there was an old softened area, and in the frontal lobe a focus having a granular, or pitted, appearance.

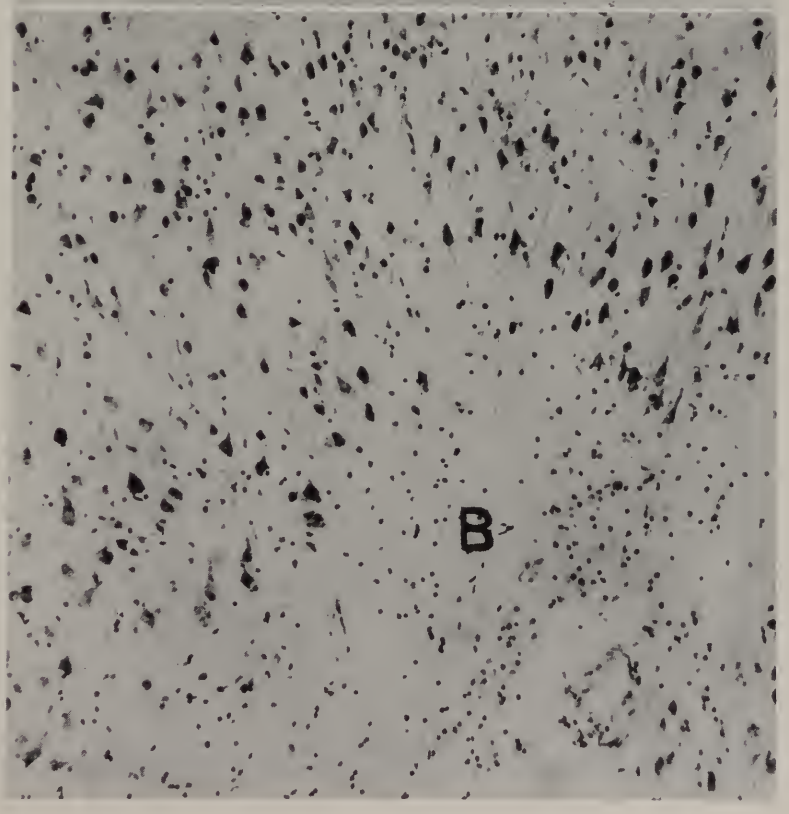
Microscopically the surface of the brain shows numerous shallow indentations. Beneath the depressions there are small scars devoid of nerve cells (A). Characteristic of this condition is the absence of cavity formation. The small round cells (B) scattered through the scars were found with silver stains to be glial in nature. The "protruding" and even parts of the cortex are relatively normal.

This is one of the more uncommon pathologic changes in cerebral arteriosclerosis. The scars are thought to represent old infarcts. The condition is also referred to as "granular atrophy" of the cerebral cortex.

Reference: Hassin, G. B.: Histopathology of the peripheral and central nervous systems, ed. 2, New York, Hoeber, 1940, p. 330.

SLIDE 18. VERRUCOUS ATROPHY OF THE CEREBRAL CORTEX





NEG. 72758 X 15 NEG. 72761 X 150

TRAUMATIC SOFTENING OF THE BRAIN, OF TEN DAYS' DURATION

Hematoxylin and Eosin Stain

CLINICAL NOTE: A 14-year-old boy who fell off his bicycle. He was unconscious for ten days when death supervened. (A 3195)

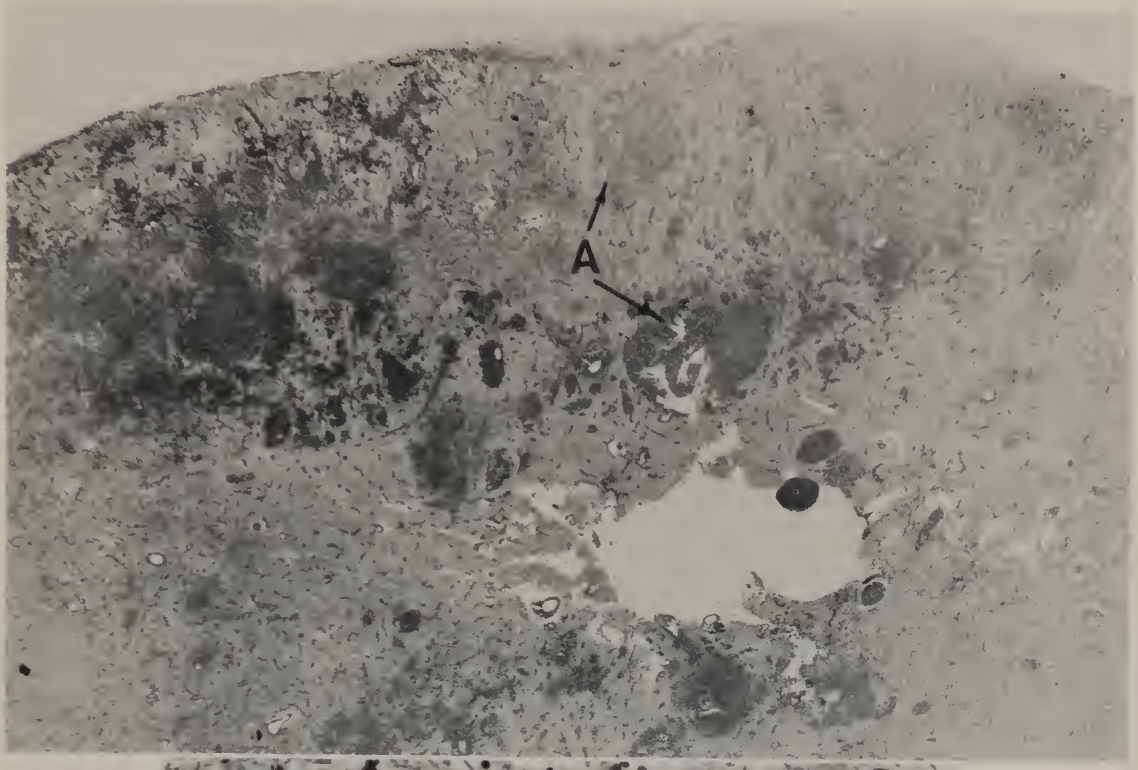
PATHOLOGY: Fractures were found in the right middle and posterior cranial fossae. There was a small subdural hematoma. The left temporal lobe was contused. Both the leptomeninges and the cerebrum were congested.

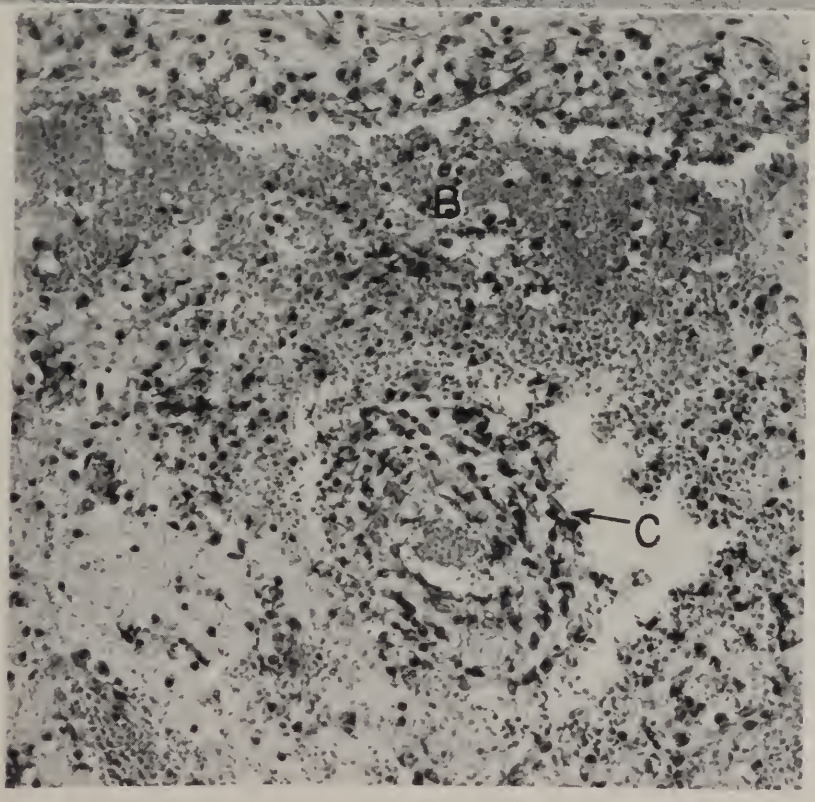
The section has been taken from the contused temporal lobe. It shows necrotic areas containing multiple hemorrhages (A). There is diffuse subpial hemorrhage (B) with reactive cells in the overlying leptomeninges. Penetrating cortical vessels are hyperplastic and edematous (C). New vessel formation is prominent. Gitter cells are in abundance.

Parts of the brain adjacent to the necrotic areas contain many perivascular hemorrhages. In some of these are small round cells and numerous gitter cells, some of which contain ingested pigment. The brownish black pigment seen particularly in the hemorrhagic zones is an artefact produced by fixation with formalin.

Reference: Rand, C. W., and Courville, C. B.: Histologic studies of the brain in cases of fatal injury to the head, Arch. Neurol. & Psychiat. 36: 1277, 1936.

SLIDE 20. TRAUMATIC SOFTENING OF THE BRAIN, OF TEN DAYS' DURATION





NEG. 72760 X 15 NEG. 73983 X 205

FAT EMBOLISM OF THE BRAIN

Fat Stain

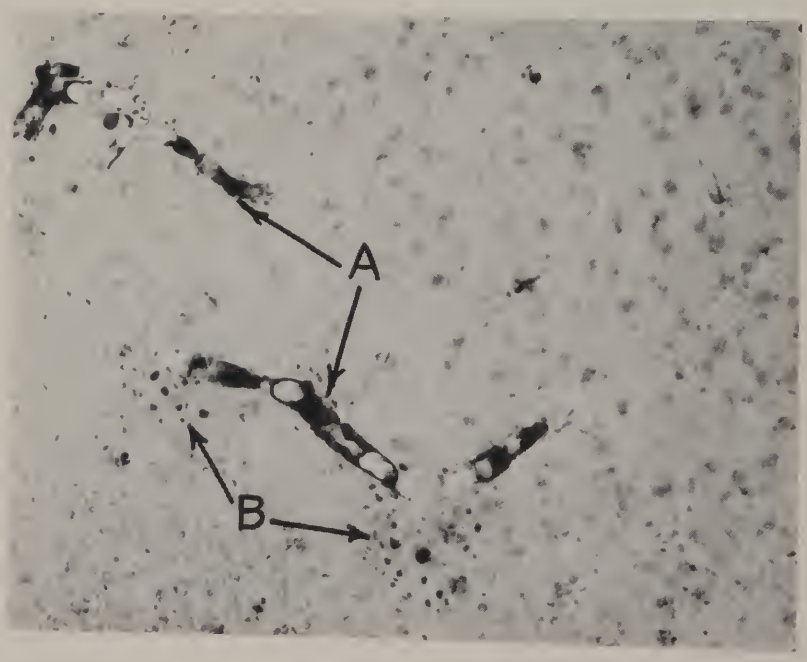
CLINICAL NOTE: Data not available.

PATHOLOGY: The section shows many capillaries plugged by fat emboli (A). In some regions one finds the globules free in the parenchyma (B). Here and there early phagocytosis of the fat is in progress.

Fat embolism occurs most frequently after fracture of long bones and after crush injuries to parts of the body heavily clothed with adipose tissue. Frequently there are associated punctate hemorrhages of the brain, especially in the white matter.

Reference: Vance, B. M.: The clinical diagnosis of fat embolism, Am. J. Surg. 26: 27, 1934.

Gonzales, T. A., Vance, M., and Helpern, M.: Legal medicine and toxicology, New York, D. Appleton-Century Co., 1940, pp. 88-102.



NEG. 72731 X 150

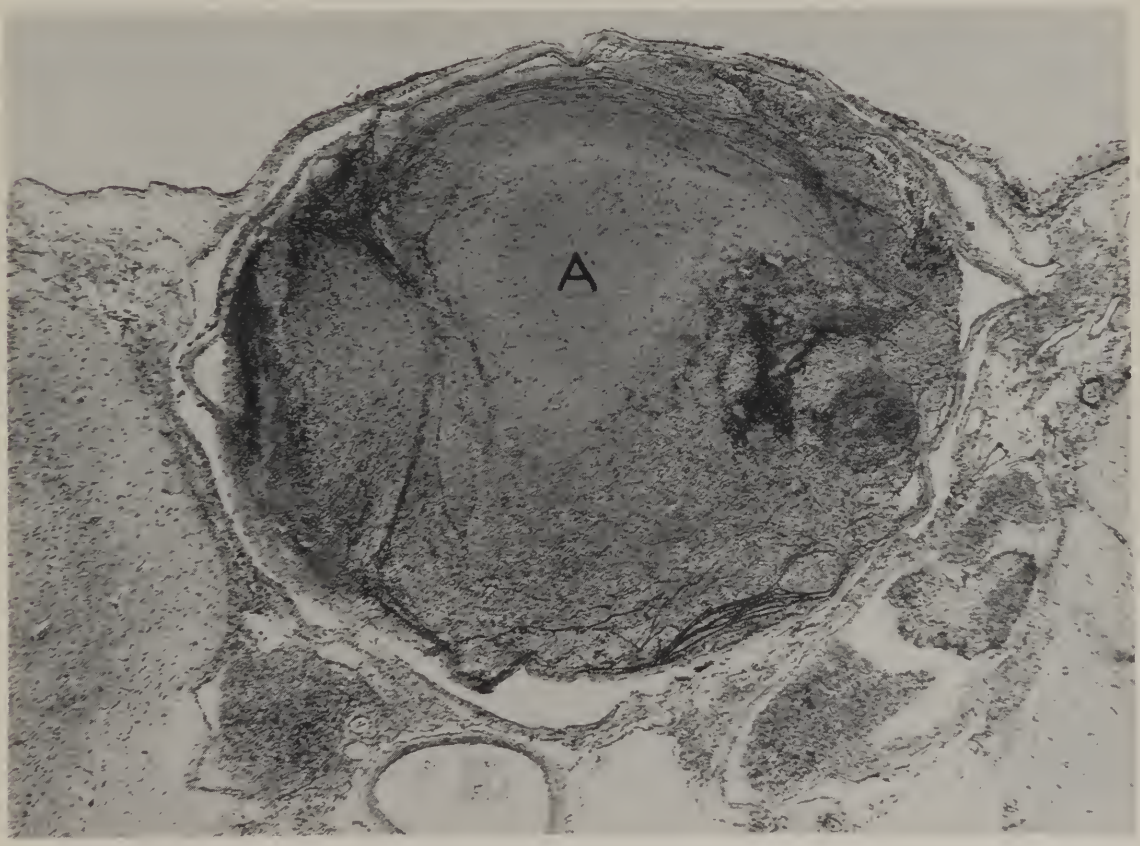
THROMBOSIS OF LEPTOMENINGEAL VEIN SECONDARY TO
THROMBOSIS OF THE SAGITTAL SINUS
Hematoxylin and Eosin Stain

CLINICAL NOTE: From same case as Slide 9. (A 3361)

PATHOLOGY: The changes present in this section are similar to those in Slide 8. Outstanding is a large pial vein containing an organized thrombus (A). In the meshes of the adjacent pia-arachnoid there are numerous actively phagocytosing histiocytes.

The adjacent cortex is edematous, and contains small hemorrhages, some of which are perivascular.

Reference: Doyle, J. B.: Obstruction of the longitudinal sinus, Arch. Neurol. & Psychiat. 18: 374, 1927.



NEG. 72741 X 30

CARBON MONOXIDE POISONING

Hematoxylin and Eosin Stain

CLINICAL NOTE: A 61-year-old man inhaled carbon monoxide in an attempt at suicide. He died the following day. (NP 128)

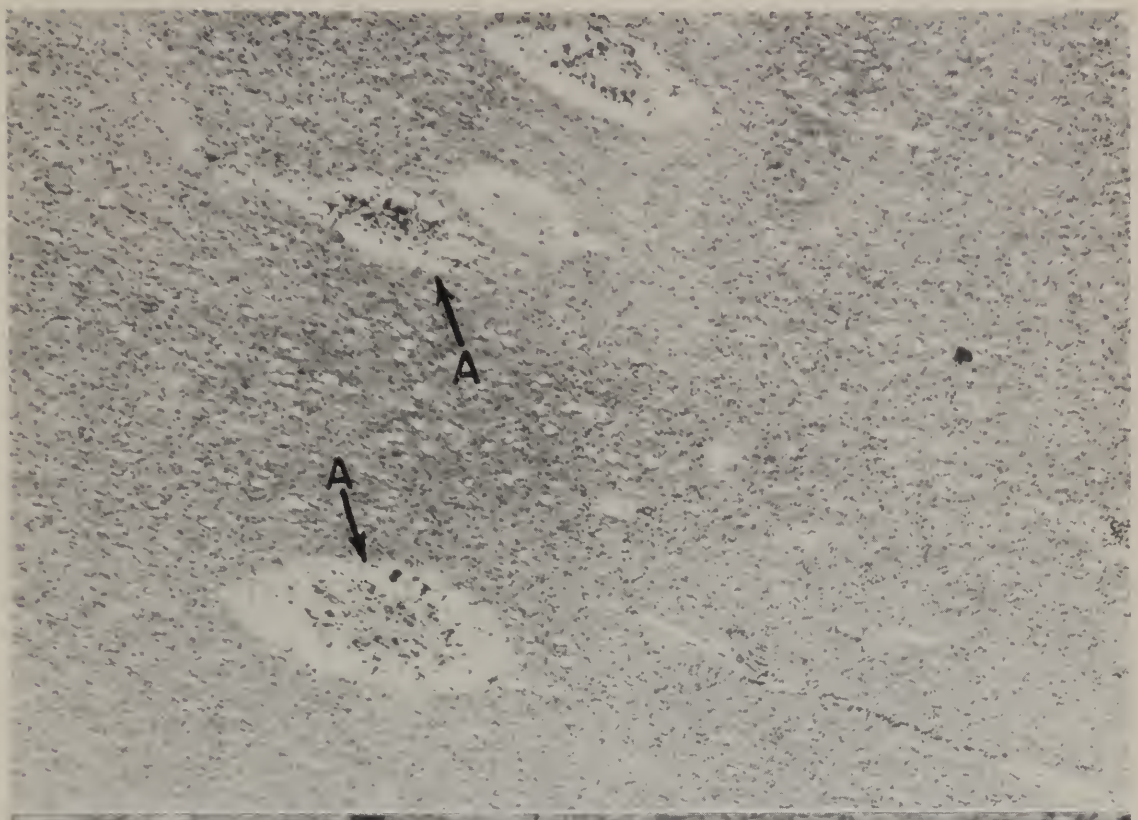
PATHOLOGY: An early bronchopneumonia was found. The meningeal vessels were intensely congested. On section the white matter was found to contain numerous petechiae.

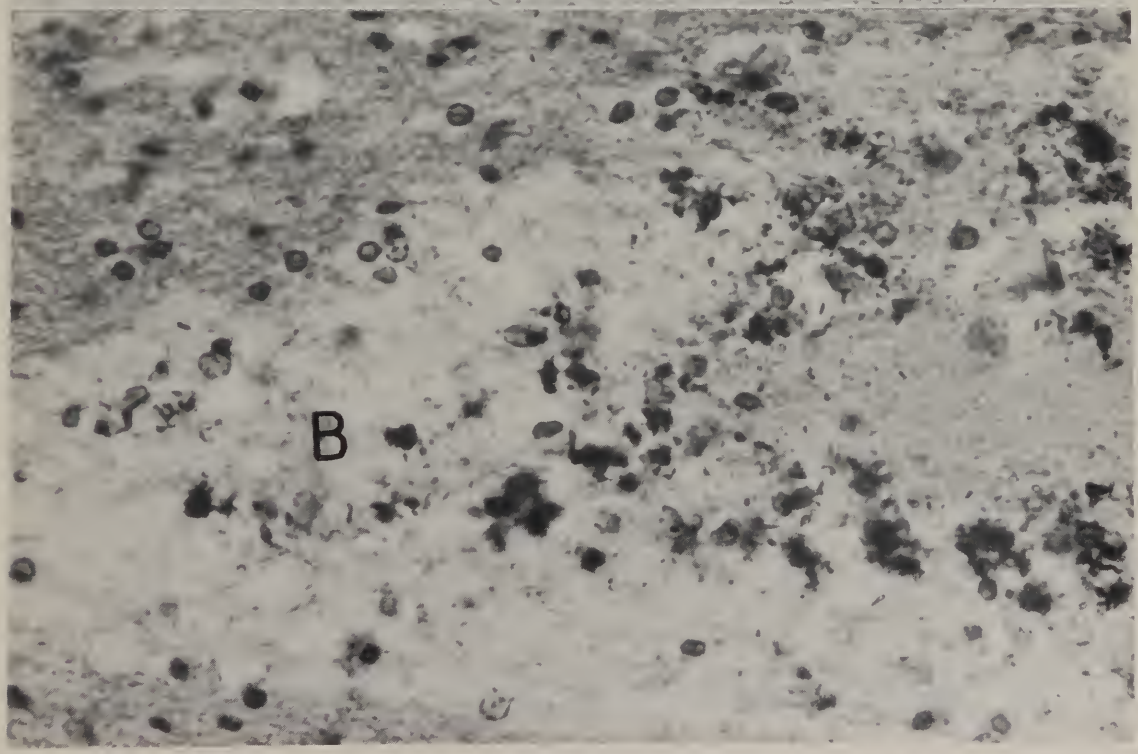
The leptomeninges are greatly edematous and congested, and the arachnoid meshes are strewn with erythrocytes. Except for moderate edema no changes are noted in the cerebral cortex. In the underlying white matter, however, one finds well demarcated foci of pale spongy tissue from which myelin has disappeared (A). Closer examination shows that the foci are packed with erythrocytes (B). There are occasional ring hemorrhages with necrotic centers and "palisading" glia cells. The pigment present in the demyelinated areas is formalin artefact.

Changes in the globus pallidus in carbon monoxide poisoning are shown in Slide 38.

Reference: Hsu, Y. K., and Chang, Y. L.: Cerebral subcortical myelinopathy in carbon monoxide poisoning, Brain 61: 384, 1938.

SLIDE 23. CARBON MONOXIDE POISONING





NEG. 72744a X 65 NEG. 72744b X 705

ACUTE MENINGITIS

Nissl Stain

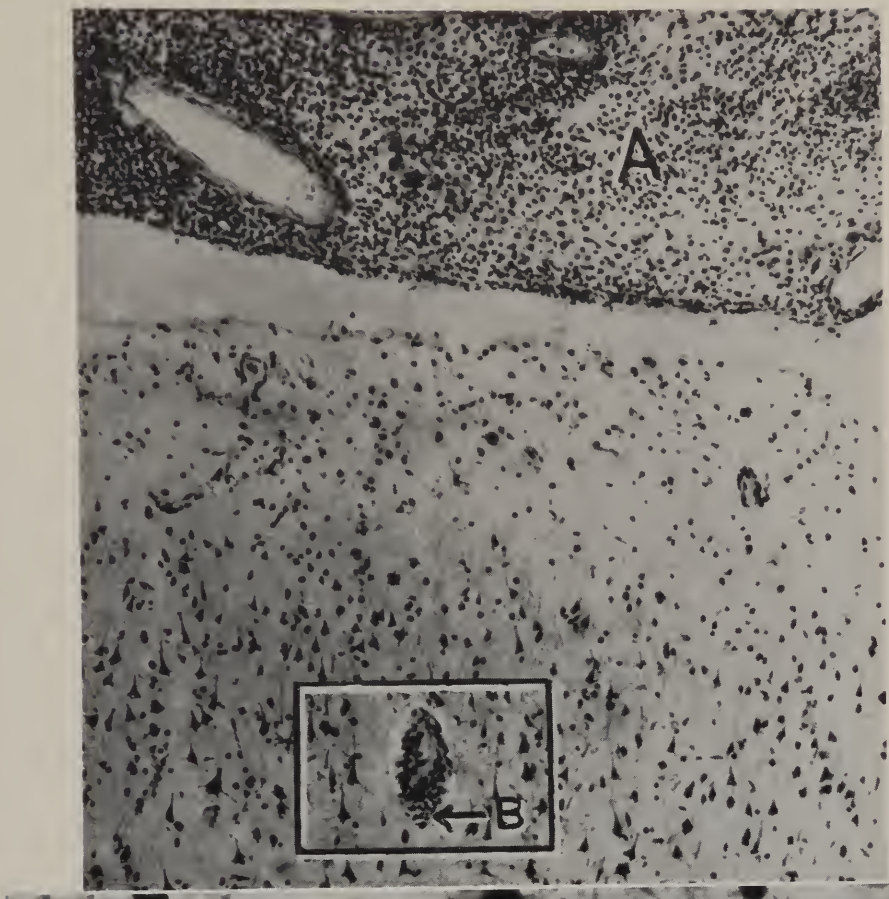
CLINICAL NOTE: A 66-year-old male with otitis media who died of an acute meningitis. (A 3554)

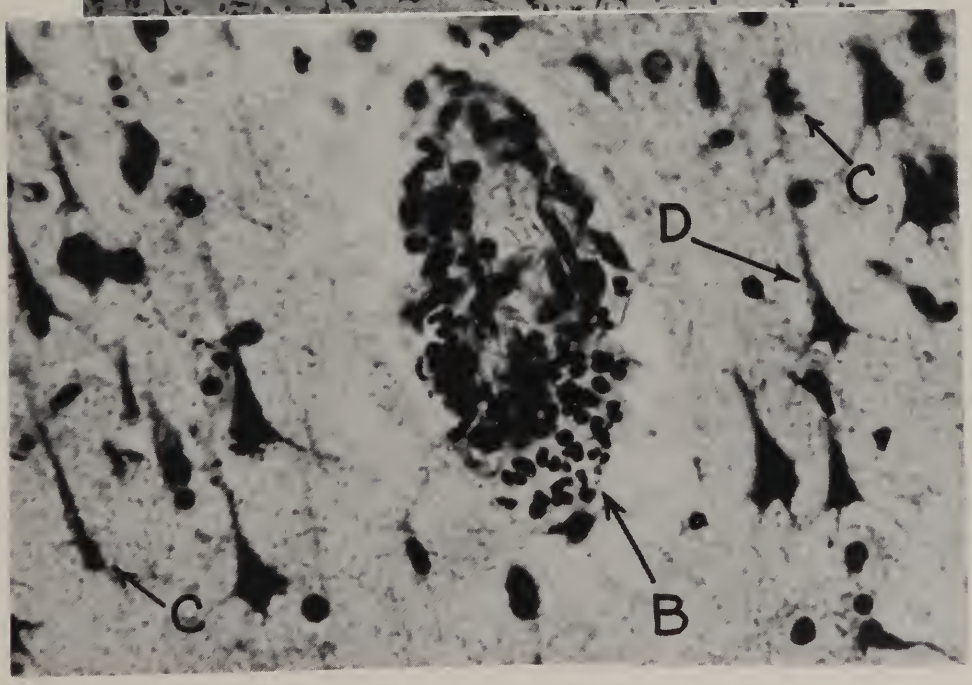
PATHOLOGY: The section shows in the leptomeninges (A) an exudate consisting chiefly of polymorphonuclear leucocytes. Numerous lymphocytes and plasma cells are also noted. The inflammatory exudate is continuous downward in the perivascular spaces of penetrating cortical vessels (B).

The cortex adjoining the pia-arachnoid exhibits various toxic degenerative changes of ganglion cells, e.g., shrinkage of cell bodies and pyknosis of nuclei (C), transformation of cell bodies into "ghosts", and swelling and tortuosity of apical dendrites (D). Early satellitosis is visible. In the upper layers of the cortex one notes a sprinkling of polymorphonuclear leucocytes.

Reference: Brain, W. R.: Diseases of the nervous system, ed. 2, London, Oxford University Press, 1940, p. 348.

SLIDE 25. ACUTE MENINIGITIS





NEG. 74204 X 150 NEG. 73974 X 600

ACUTE MENINGITIS

Nissl Stain

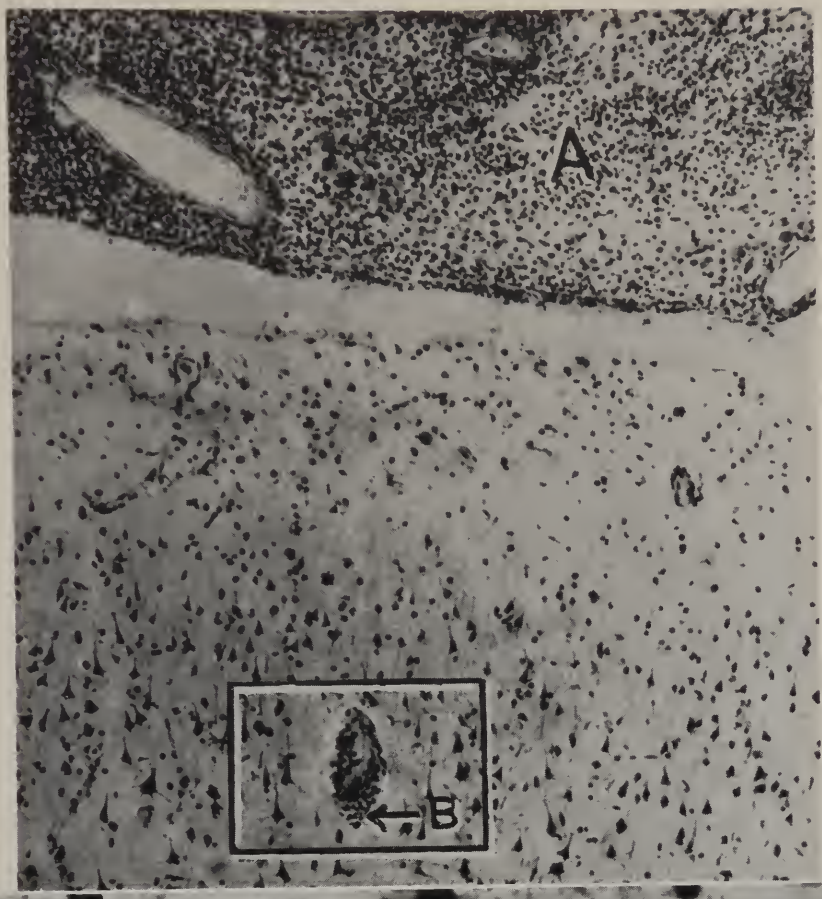
CLINICAL NOTE: A 66-year-old male with otitis media who died of an acute meningitis. (A 3554)

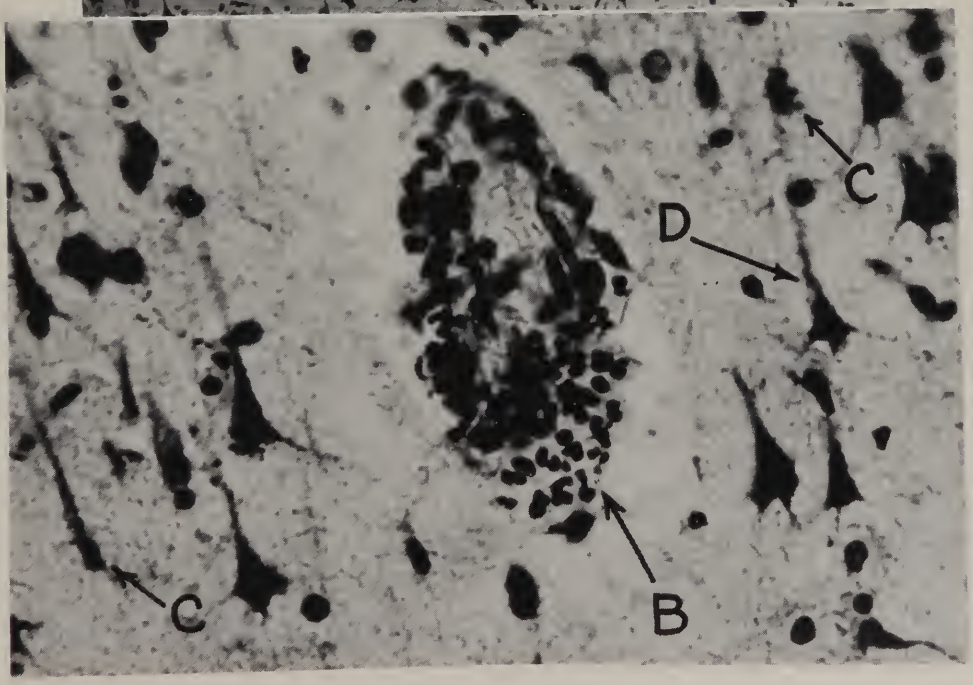
PATHOLOGY: The section shows in the leptomeninges (A) an exudate consisting chiefly of polymorphonuclear leucocytes. Numerous lymphocytes and plasma cells are also noted. The inflammatory exudate is continuous downward in the perivascular spaces of penetrating cortical vessels (B).

The cortex adjoining the pia-arachnoid exhibits various toxic degenerative changes of ganglion cells, e.g., shrinkage of cell bodies and pyknosis of nuclei (C), transformation of cell bodies into "ghosts", and swelling and tortuosity of apical dendrites (D). Early satellitosis is visible. In the upper layers of the cortex one notes a sprinkling of polymorphonuclear leucocytes.

Reference: Brain, W. R.: Diseases of the nervous system, ed. 2, London, Oxford University Press, 1940, p. 348.

SLIDE 25. ACUTE MENINIGITIS





NEG. 74204 X 150 NEG. 73974 X 600

GENERAL PARESIS (TREATED)

Nissl Stain

CLINICAL NOTE: A 44-year-old woman who showed symptoms of general paresis about six months prior to death. Malarial therapy was employed (number of chills not recorded) and then later she received arsphenamine. The ensuing arsenic dermatitis, abscesses and enteritis proved fatal. (A 3449)

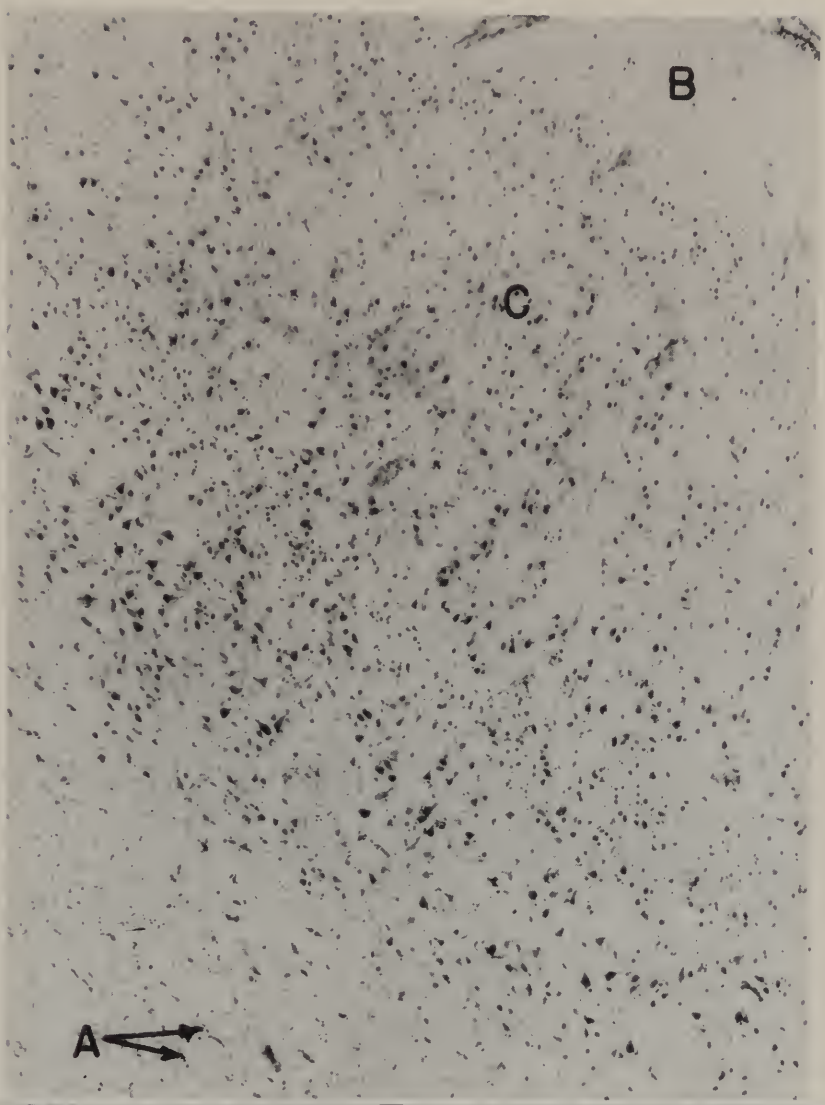
PATHOLOGY: Autopsy confirmed the clinical diagnoses. Moderate syphilitic aortitis was found. The brain weighed 1250 grams. Grossly it seemed normal.

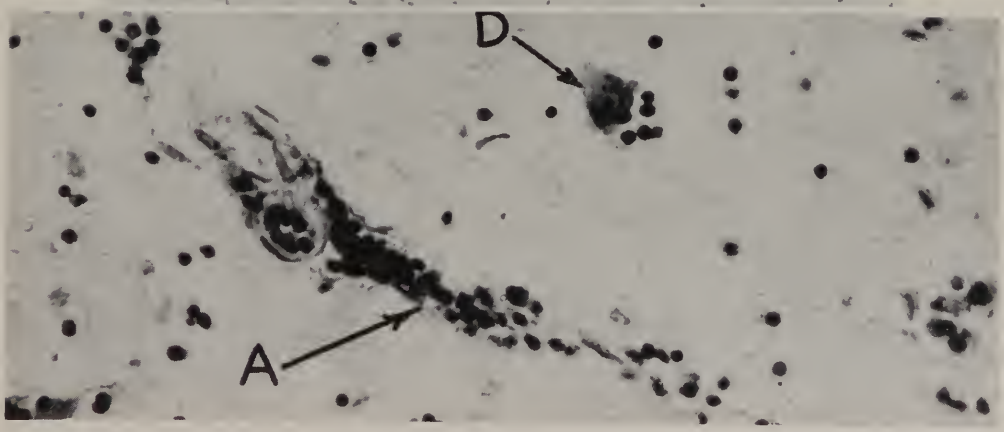
The section is taken from the hippocampal formation and adjoining temporal gyri. The leptomeninges of the temporal cortex show moderate to scanty diffuse infiltration with lymphocytes and plasma cells. Similar cells are present in the perivascular spaces of the cortex and white matter (A). A slight decrease in the number of ganglion cells is noted in the molecular layer (B) and in the layers of small and medium sized pyramidal cells (C). In these regions, many ganglion cells are in various stages of degeneration, some being transformed into ghosts. Glial cells are, in general, scanty but in the lower parts of the cortex one notes well advanced neuronophagia (D). Similar changes are present in the pyramidal layer of the hippocampus. Here one finds newly-formed blood vessels.

Because of the relative mildness of the changes it seems logical to assume that the malarial therapy may have been effective in decreasing a pre-existent marked inflammatory reaction.

Reference: Ferraro, A.: The pathology of paresis after treatment with malaria, Arch. Neurol. & Psychiat. 21: 69, 1929.

SLIDE 26. GENERAL PARESIS (TREATED)





NEG. 72739 X 65 NEG. 73981 X 450

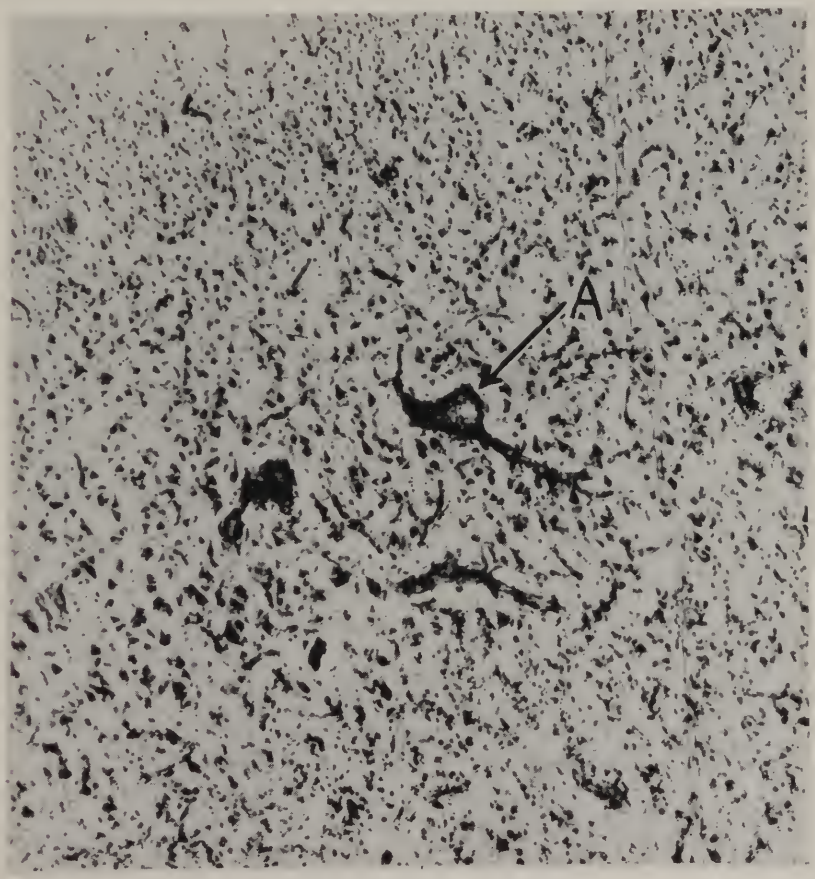
GENERAL PARESIS (UNTREATED)

Nissl Stain

CLINICAL NOTE: Data not available.

PATHOLOGY: The part of the cortex submitted is not designated. The changes are similar to those in the previous slide, but are considerably more marked. The cortical architecture is indistinct. Many of the ganglion cells are pale. A moderate number of rod cells are scattered throughout the cortex. Many lymphocytes and plasma cells are present in the leptomeninges as well as in perivascular spaces of the cortex (A) and the white matter.

Reference: Galbraith, A. J.: Some problems in the histopathology of general paralysis of the insane, Brit. J. Ven. Dis. 14: 197, 1938.



NEG. 72757 X 70

FOCAL EMBOLIC ENCEPHALITIS AND MENINGITIS COMPLICATING SUBACUTE BACTERIAL ENDOCARDITIS

Hematoxylin and Eosin Stain

CLINICAL NOTE: This is a case of a 30-year-old woman who had subacute bacterial endocarditis with symptoms indicating cerebral involvement. (A 3171)

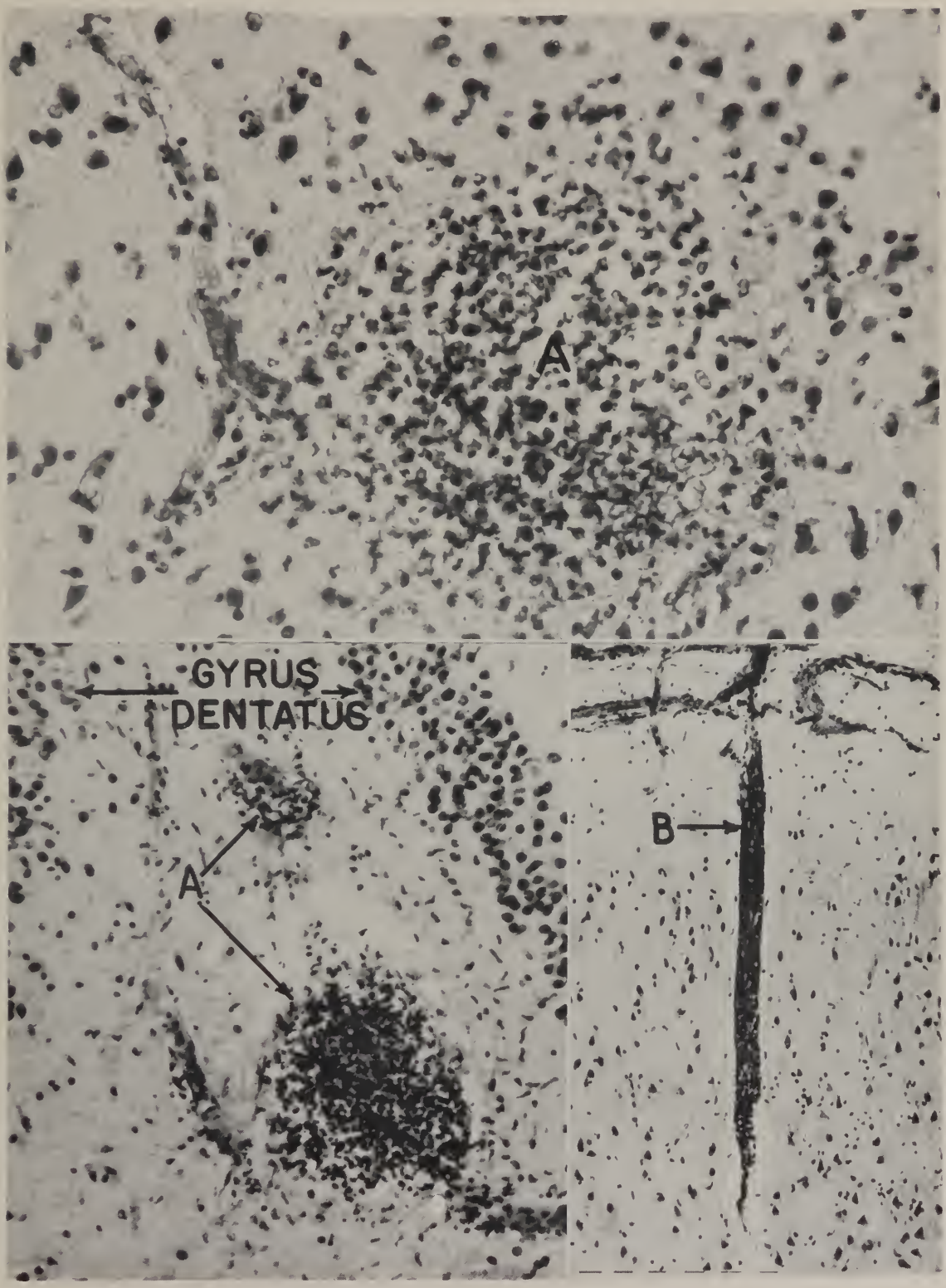
PATHOLOGY: The section is taken from the hippocampal formation and adjoining temporal gyri. Scattered throughout the cortex, and to a lesser degree the white matter, are so-called "glial nodules" (A) the result probably of a shower of minute emboli. Virtually all the nodules are adjacent to blood vessels. In some of the foci, such as the ones photographed, there are numerous round cells and some polymorphonuclear leukocytes. Both endothelium and adventitia of the involved blood vessels are proliferated.

There is also a leptomeningitis, of moderate severity, the predominant cells being lymphocytes, plasma cells and histiocytes. Cortical penetrating vessels (B) are surrounded by similar cells.

This is one of several histopathologic pictures encountered in bacterial endocarditis.

Reference: Winkelman, N. W., and Eckel, J. L.: The brain in bacterial endocarditis, Arch. Neurol. & Psychiat. 23: 1161, 1930.

SLIDE 28. FOCAL EMBOLIC ENCEPHALITIS AND MENINGITIS COMPLICATING SUBACUTE BACTERIAL ENDOCARDITIS



NEG. 72732 X 230 NEG. 74201 X 390 NEG. 74202 X 150

BRAIN ABSCESS

Hematoxylin and Eosin Stain

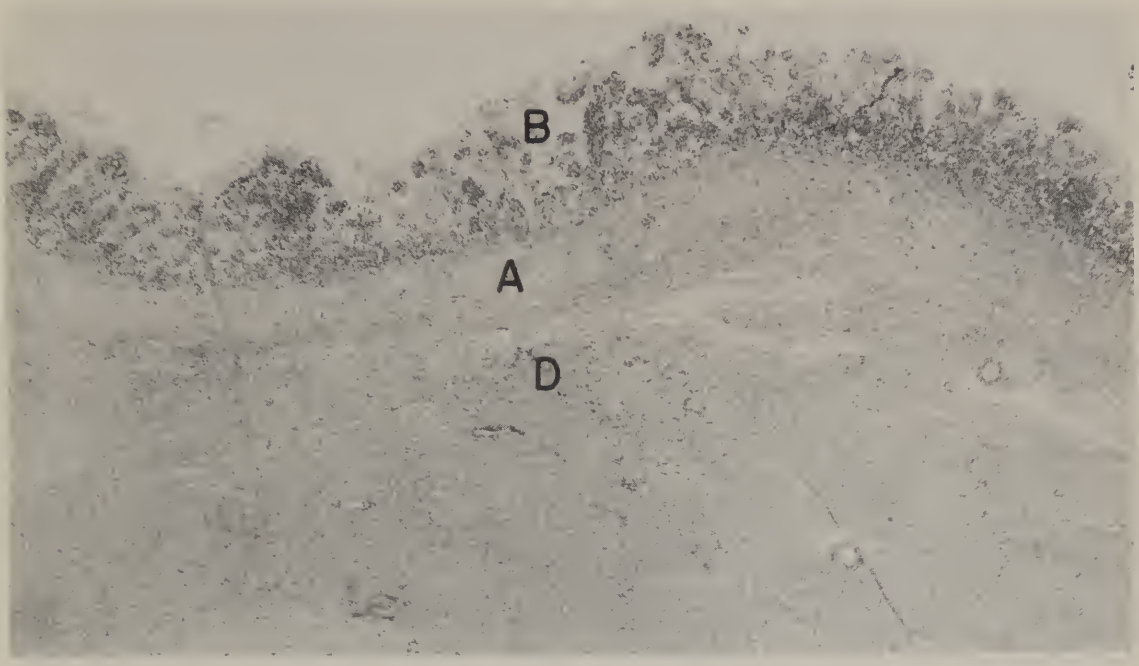
CLINICAL NOTE: The case of a 17-year-old male who had a nasal infection which was complicated by osteomyelitis of the skull and brain abscess. (A 3266)

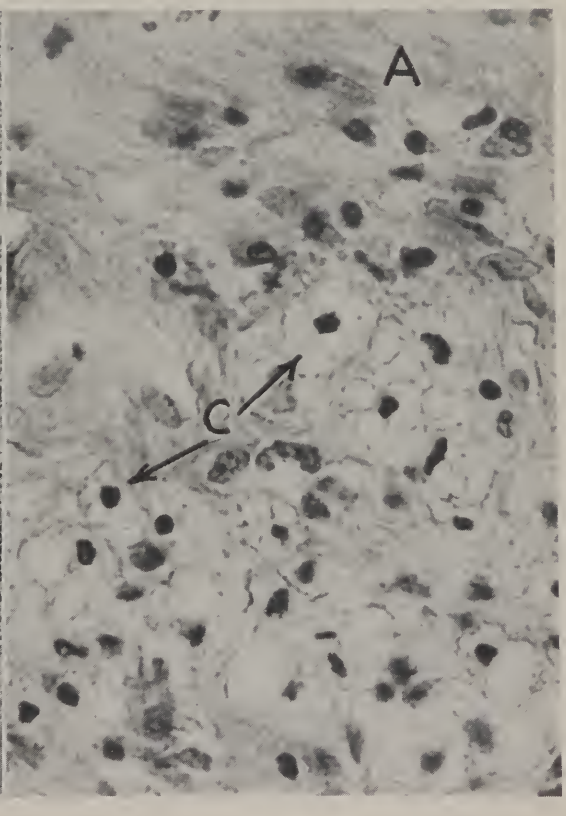
PATHOLOGY: The abscess is surrounded by a thick fibrous "capsule" (A). The part of the core remaining (B) consists of collections of polymorphonuclear leucocytes, lymphocytes and plasma cells. In the marginal zone between the purulent exudate and "capsule" there are numerous gitter cells (C).

The brain substance (D) adjacent to the abscess contains many astrocytes.

Reference: Freeman, W.: Neuropathology. The anatomical foundation of nervous diseases, Philadelphia, Saunders, 1933.

SLIDE 29. BRAIN ABSCESS





NEG. 73975 X 200 NEG. 72730a X 50 NEG. 72730b X 705

MENINGITIS IN SYPHILITIC INFANT

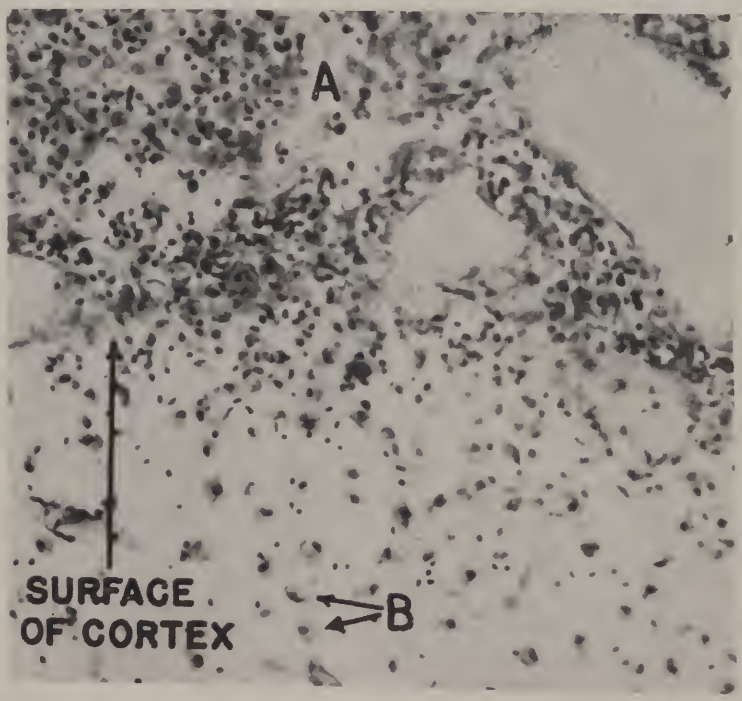
Nissl Stain

CLINICAL NOTE: A boy, aged 2, who during the last few months of life had had increasing stiffness of the extremities. On admission, the lower extremities were found to be spastic. A maculo-papular rash covered the face and scalp. Spinal fluid Wassermann, 4 plus; protein, 70 mg. per cent; sugar, 58 mg. per cent. Weakness increased and the child died four days after admission to hospital. (A 2660)

PATHOLOGY: The leptomeninges are greatly thickened and congested (A). The meshes are markedly infiltrated by round cells and macrophages. One notes more polymorphonuclear leukocytes than are usually present in syphilis.

In some parts of the subjacent cortex there is considerable wiping out of ganglion cells with replacement by various glial elements, such as gitter cells (B) and rod cells. Marked hyperemia is noted in both cortex and white matter.

Reference: Bly, P. A.: Syphilitic meningitis in infants and young children, with a report of a case, Am. J. Syph. 2: 712, 1918.



Slide 30 NEG. 72740 X 250

PERIVENOUS ENCEPHALITIS

Nissl Stain

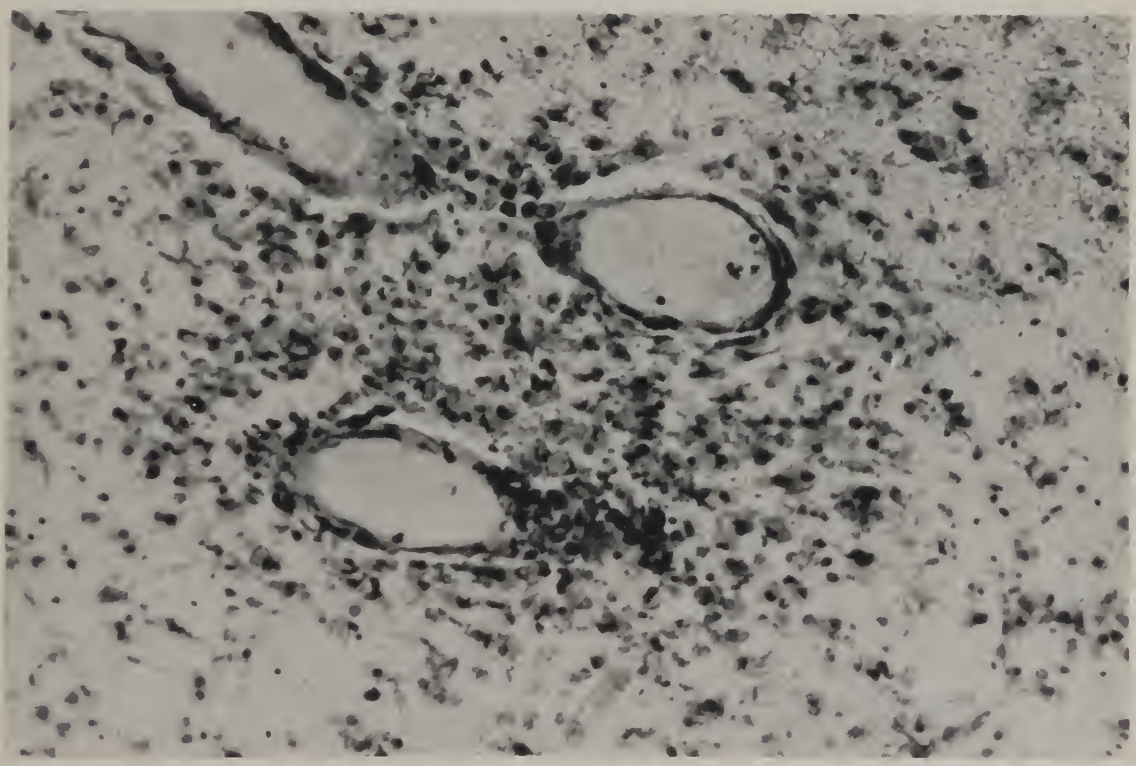
CLINICAL NOTE: Not available. (NP 155)

PATHOLOGY: The inflammatory changes are most pronounced in the white matter. Here some of the veins are surrounded by accumulations of cells. Differential stains show these to be mostly microglia and oligodendroglia. Such a focus is shown in the photograph.

The leptomeninges are somewhat thickened and in some parts of the subpial cortex one notes reactive astrocytosis.

The pathologic changes do not throw any light on the nature of of the causative agent.

Reference: Wilson, S. A. K.: Neurology, Baltimore, Williams & Wilkins, 1940, vol. 1, p. 53.



NEG. 72735 X 330

Slide 31

MIDBRAIN IN EPIDEMIC ENCEPHALITIS

Nissl Stain

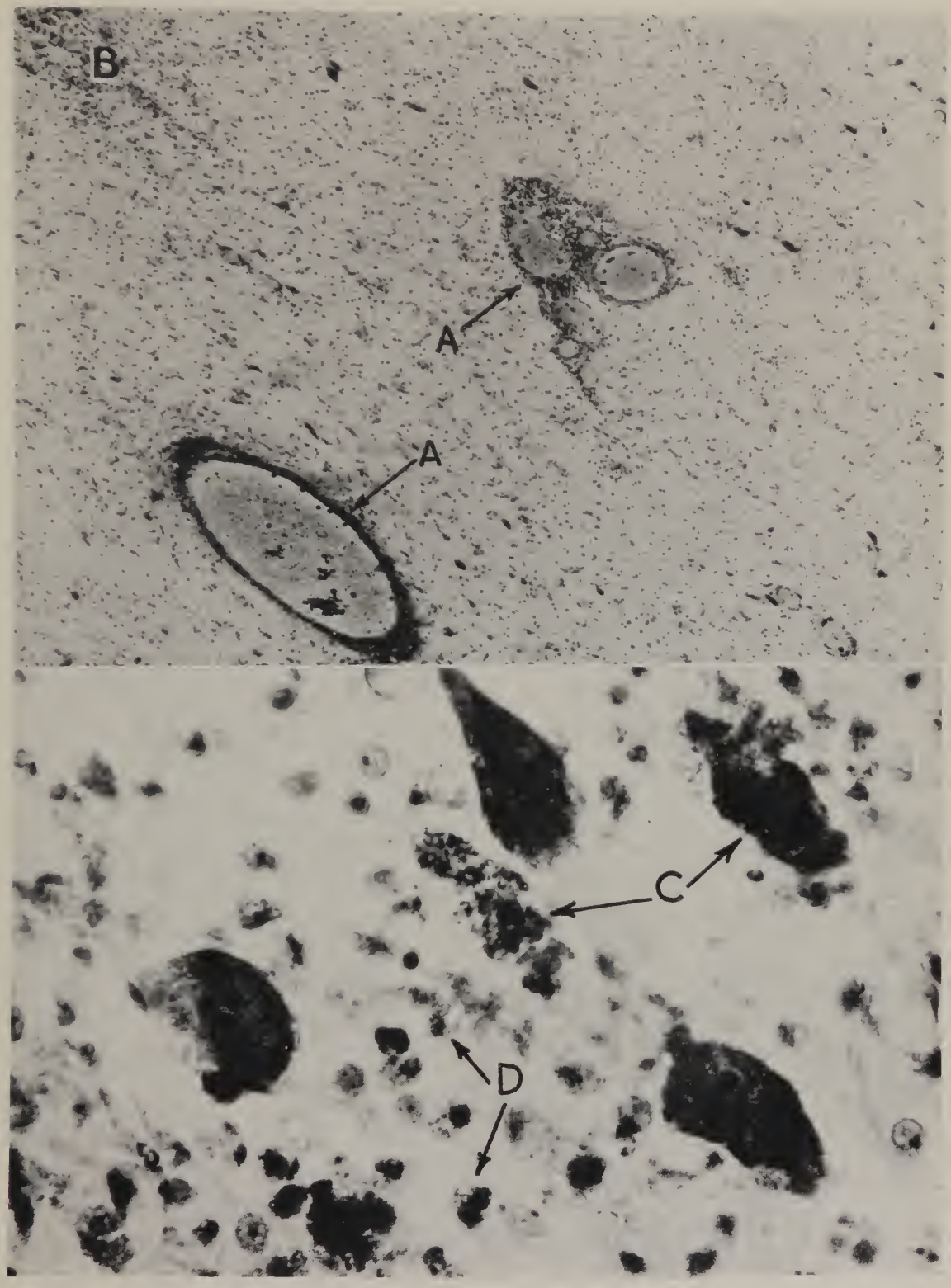
CLINICAL NOTE: A 30-year-old male who developed acute epidemic encephalitis. About 10 days later he died of bronchopneumonia. (A 2896)

PATHOLOGY: The section is through the level of the substantia nigra. There is marked perivascular (particularly perivenous) infiltration with round cells (A). Several glial nodules (B), consisting chiefly of proliferated microglia, are observed. Neuroglial and microglial cells are scattered diffusely throughout the section but tend to be accumulated in the vicinity of the melanin-containing cells. Here and there such cells have undergone disintegration (C), their pigment being taken up by microglial cells (D). "Formalin pigment", an artefact, is present in and around the larger vessels.

The substantia nigra is one of the chief sites of involvement in epidemic encephalitis.

Reference: Bassoe, P., and Hassin, G. B.: A contribution to the histopathology of epidemic ("lethargic") encephalitis, Arch. Neurol. & Psychiat. 2: 24, 1929.

SLIDE 32. MIDBRAIN IN EPIDEMIC ENCEPHALITIS



NEG. 72743a X 70 NEG. 72743b X 810

ARTERIAL THROMBOSIS

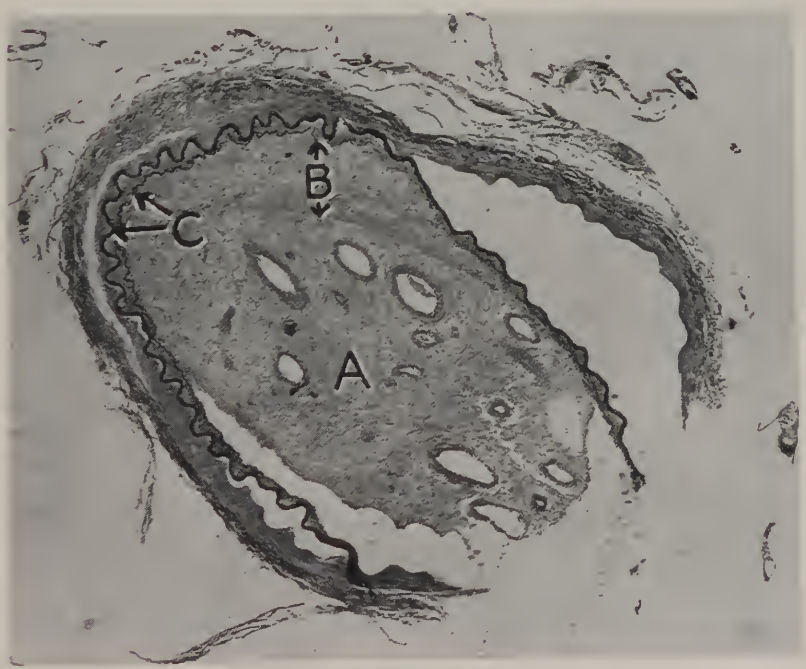
Van Giesen Stain

CLINICAL NOTE: Same case as in Slide 30. (A case of syphilis) (A 2660)

PATHOLOGY: The lumen of the artery is filled with a cylindrical mass of sparsely cellular fibrous tissue (A). The appearances indicate that most if not all of this mass is a well-organized, old, recanalized thrombus. The part adjacent to the internal elastic lamina may represent proliferated intima (B), such as one sees in Heubner's syphilitic endarteritis as well as in non-syphilitic thromboses. There is a duplication of the internal elastic lamina (C). The adventitia contains a few round cells, but not as many as one usually encounters in active syphilis.

The changes as a whole are doubtless to be ascribed to syphilis but there is a considerable question that this is a case of Heubner's syphilitic endarteritis.

Reference: Jakob, A.: Anatomie und Histologie des Gehirns. In Handbuch der Psychiatrie, 1. Abteil., 1 Teil, II Band, Leipzig, Deuticke, 1929, pp. 564-568.



Slide 33 NEG. 72742 X 30

TUBERCULOUS MENINGITIS

Nissl Stain

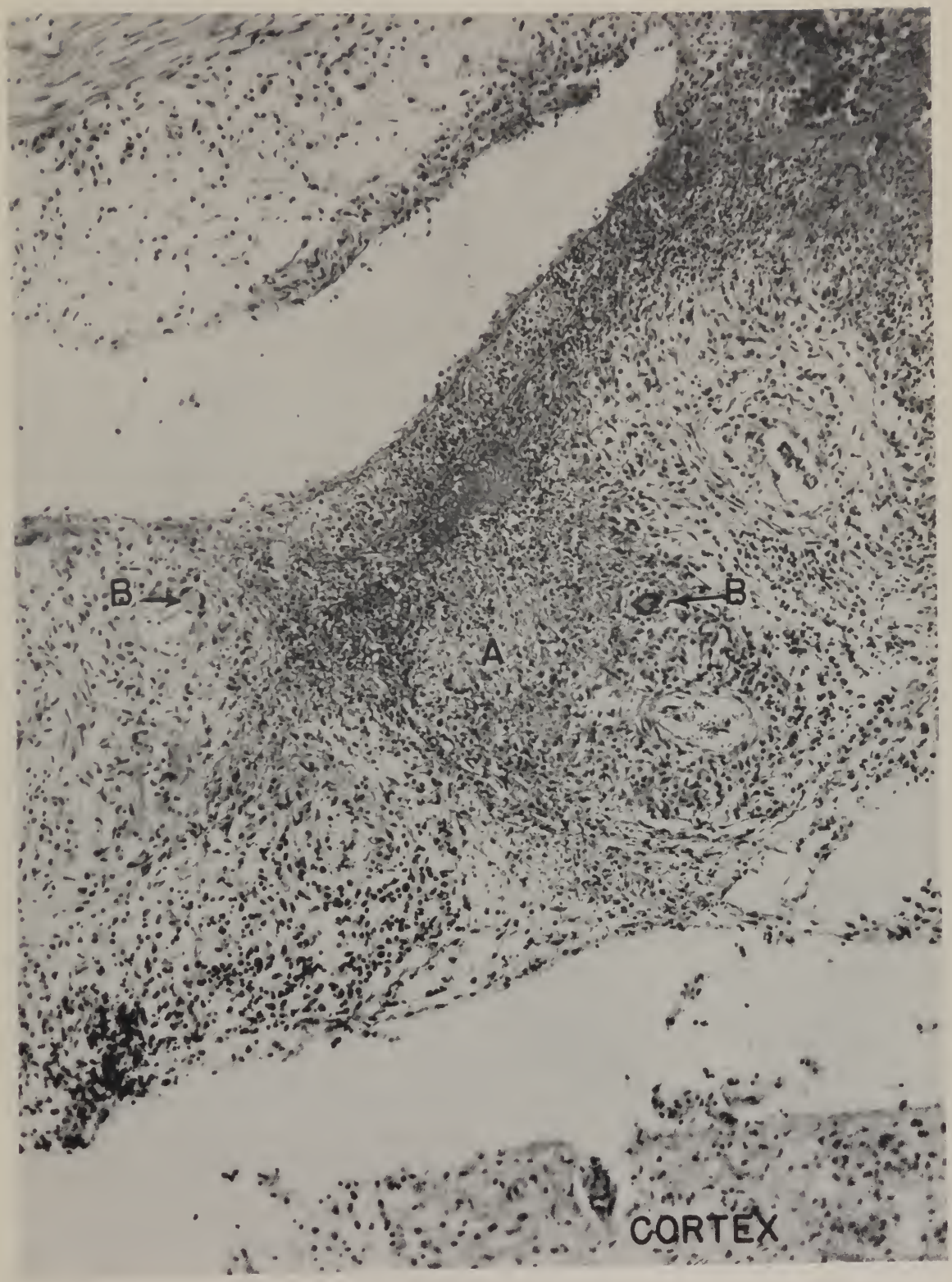
CLINICAL NOTE: A baby boy, 14 months old, was admitted to hospital in a state of lethargy and somnolence and was found to have high fever and rigidity of the neck. (A 3757)

PATHOLOGY: Autopsy disclosed miliary pulmonary tuberculosis together with meningitis. The section shows widespread changes characteristic of tuberculous meningitis. The meshes of the pia-arachnoid are widely distended. Tubercles are at various stages of formation; most of them show necrotic centers (A) in the vicinity of which are epithelioid cells, lymphocytes and plasma cells, and a few giant cells (B) of the Langhans type.

The walls of the pial vessels are hyperplastic and are markedly infiltrated with inflammatory cells.

Reference: Weil, A.: A text-book of neuropathology, Philadelphia, Lea & Febiger, 1933, pp. 145-148.

SLIDE 34. TUBERCULOUS MENINGITIS



NEG. 74199 X 150

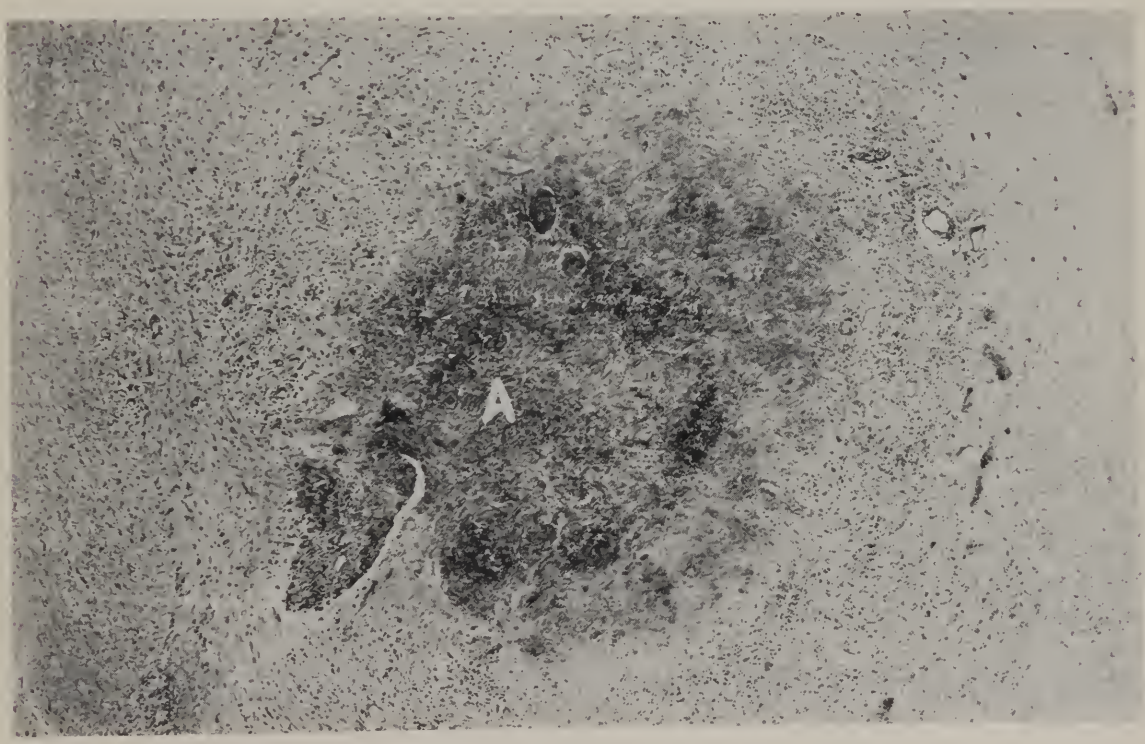
TUBERCULOMA

Hematoxylin and Eosin Stain

CLINICAL NOTE: Not available. (NP 101)

PATHOLOGY: The section shows a central core of caseous necrotic tissue containing the silhouettes of pre-existing structures (A). Fibrinous strands permeate the gumma. Surrounding the focus is a wall of granulation tissue composed of newly-formed blood vessels and accumulations of round cells, plasma cells and macrophages. The lesion merges without sharp boundary with the brain substance.

Reference: Martin, J. P.: Calcified intra-cranial tuberculomata, Brit. J. Radiol. 10: 5, 1937.



NEG. 72750 X 30

Slide 35

THE MAMILLARY BODY IN CHRONIC ALCOHOLISM

Hematoxylin and Eosin Stain

CLINICAL NOTE: Not available. (NP 199)

PATHOLOGY: Much of the mamillary body (A) is fissured and vacuolated. Perivascular edema is prominent. The scantiness of nerve cells in parts of the section indicates that some have disappeared. Those remaining are in a state of good preservation. Smaller cells are somewhat increased in number. From the appearance of their nuclei it is judged that they are mostly oligodendroglia and astrocytes. But the outstanding change is the proliferation of capillaries, which is regarded by some as the most characteristic feature of the disorder.

In the middle and dorsal regions of the hypothalamus there are perivascular hemorrhages (B). The ventricular ependyma is well preserved (C).

The condition in the mamillary body, frequently observed in chronic alcoholism, is sometimes referred to as "pseudomalacia".

Reference: Hassin, G. B.: Histopathology of the central and peripheral nervous systems, ed. 2, New York, Hoeber, 1940, pp. 270-271.

SLIDE 36. THE MAMILLARY BODY IN CHRONIC ALCOHOLISM

NEG. 74197 X 25

THE GLOBUS PALLIDUS IN CARBON MONOXIDE POISONING

Hematoxylin and Eosin Stain

CLINICAL NOTE: Not available.

PATHOLOGY: The section is a coronal one through the level of the tuberal region, the optic tract, the amygdala, the anterior thalamic nucleus, the head of the caudate nucleus, the internal capsule, the globus pallidus and the putamen.

The changes are restricted to the globus pallidus, particularly its internal division. The picture here is essentially that of encephalomalacia. Loculation is prominent. Few ganglion cells are observed and even they are pale. Glial cells, on the other hand, are greatly on the increase.

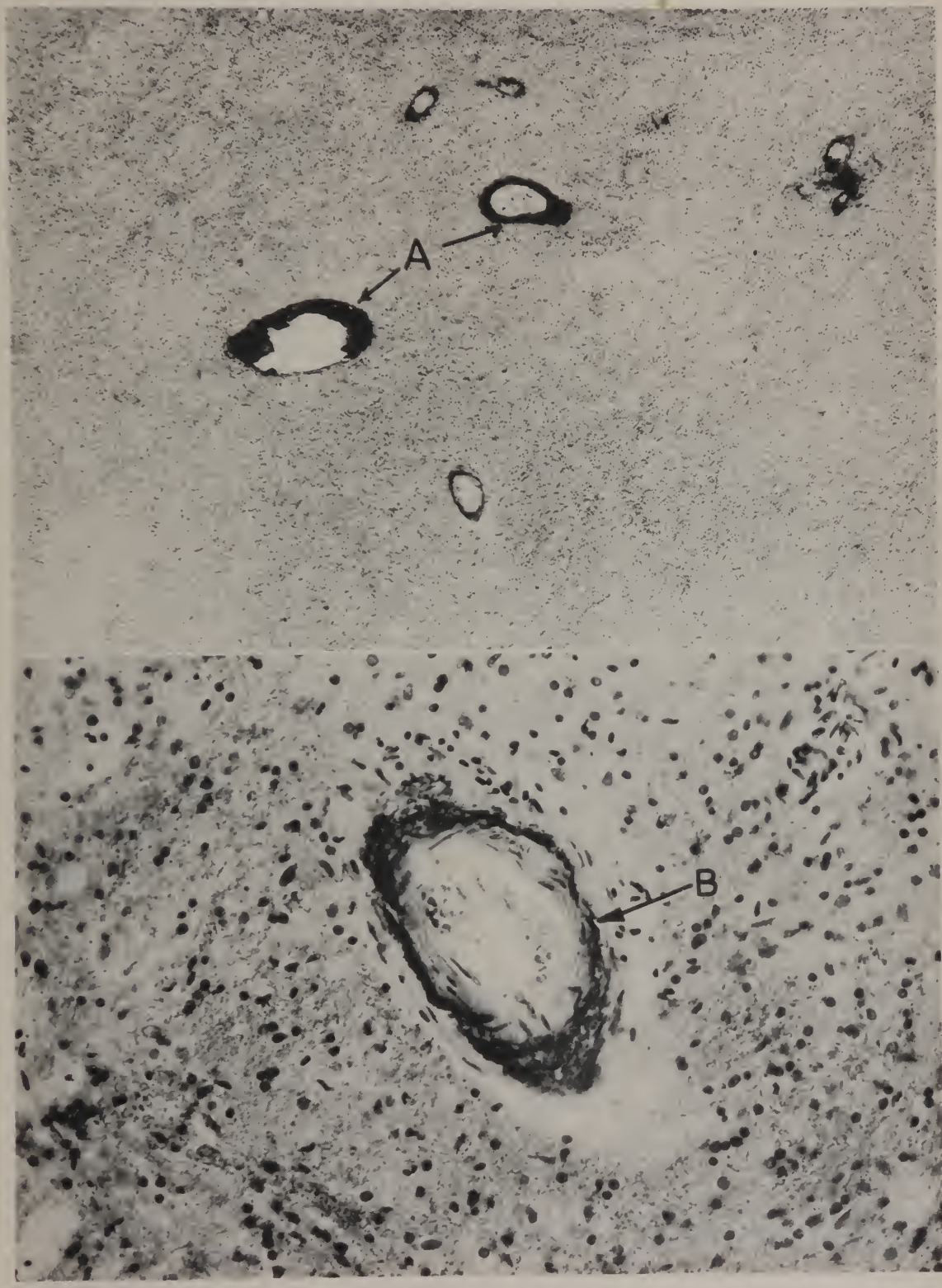
The blood vessels stand out prominently. Most of them are the seat of pseudo-calcification, or siderosis (A), which is considered normal for the globus pallidus. It has been shown that such vessel walls contain iron and a little calcium. The earliest deposition is in the media (B). In some parts of the globus pallidus one finds perivascular round cell infiltration and hemorrhage.

Changes in the subcortical white matter resulting from carbon monoxide poisoning are illustrated in Slide 23.

References: Biggart, J. H.: Pathology of the nervous system, Edinburgh, E. & S. Livingstone, 1936, pp. 74 & 75.

Semerak, C. B., and Bacon, L. H. : Experimental lesions of the brain from carbon monoxide, Arch. Path. 10: 823, 1930.

SLIDE 38. THE GLOBUS PALLIDUS IN CARBON MONOXIDE POISONING



NEG. 72773 X 90 NEG. 74200 X 350

THE CEREBELLUM IN WILSON-PSEUDOSCLEROSIS

Hematoxylin and Esoin Stain

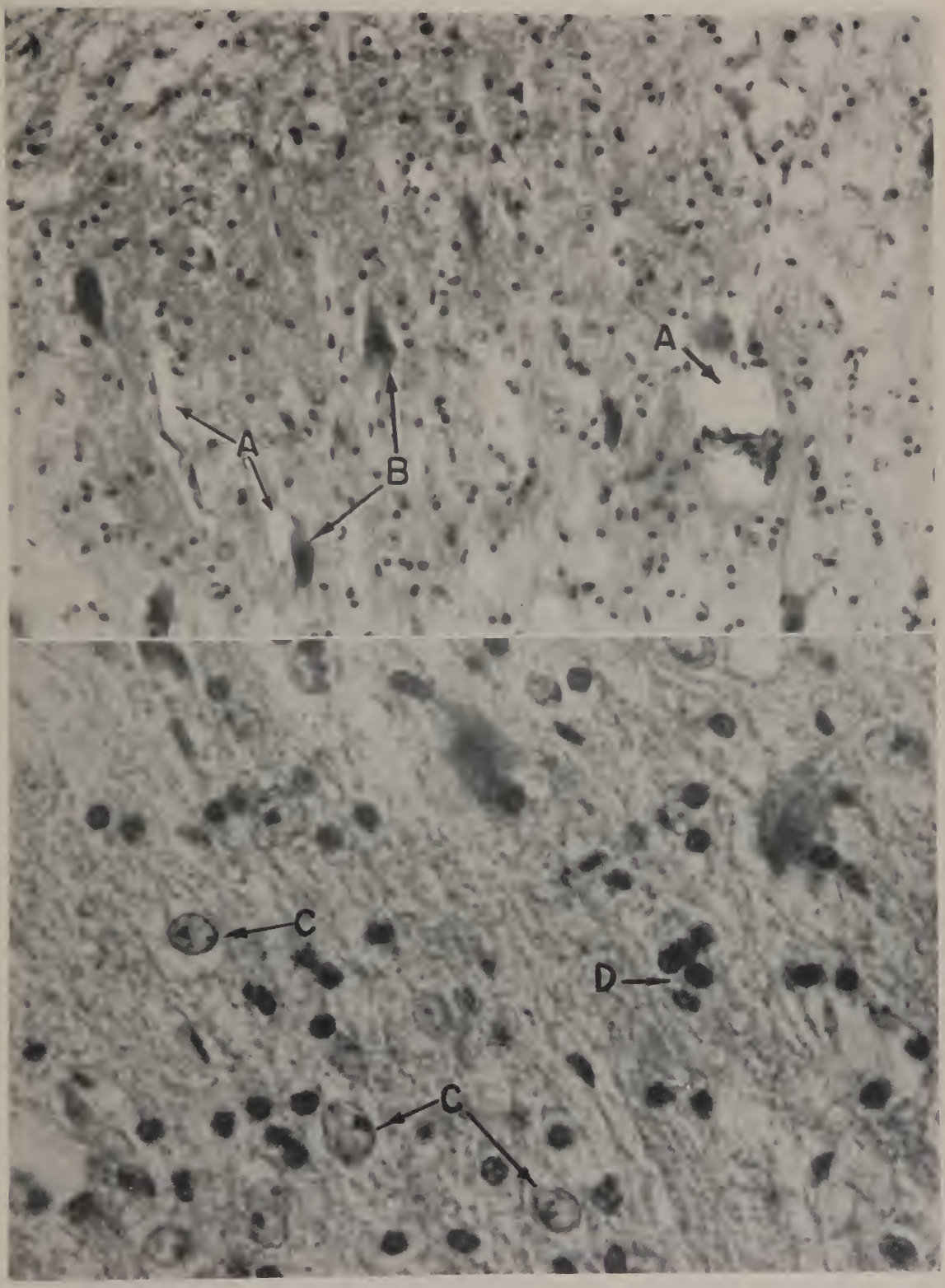
CLINICAL NOTE: A 4-year-old girl was subject to repeated attacks of depression, crying, confusion, and incoherency. There had been frequent vomiting spells and at times she was unduly drowsy. A cerebellar type of ataxia of the arms and legs was prominent. Ventriculography disclosed no changes. Terminally there were fever, pulmonary congestion, and coma. (MP 3450)

PATHOLOGY: The section is taken from the cerebellum. The dentate nucleus is edematous (A) and many of its cells are in a state of degeneration (B). Within the confines of the dentate nucleus are many large pale rounded "naked glial nuclei" (C), apparently a variety of astrocyte. The presence of such cells is characteristic but not pathognomonic of pseudosclerosis. The diffuse smaller nuclei (D) are probably those of oligodendroglia. The Purkinje cells are relatively intact; the layer of pale cells between molecular and granular layers the (so-called Bergmann layer) appears hyperplastic.

The difficulties attending the separation of Westphal-Strumpell pseudosclerosis from other forms of hepato-lenticular degeneration have been emphasized by Kinnier Wilson. Pathologically there is little to warrant subdivision. Jakob refers to the group characterized by hepato-lenticular degeneration as Wilson-pseudosclerosis.

Reference: Wilson, S. A. K.: Neurology, Baltimore, Williams & Wilkins, 1940, vol. 2, pp. 821-827.

SLIDE 39. THE CEREBELLUM IN WILSON-PSEUDOSCLEROSIS



NEG. 72736a X 320 NEG. 72736b X 925

CEREBRAL CORTEX IN HUNTINGTON'S CHOREA

Nissl Stain

CLINICAL NOTE: Not available. (NP 161)

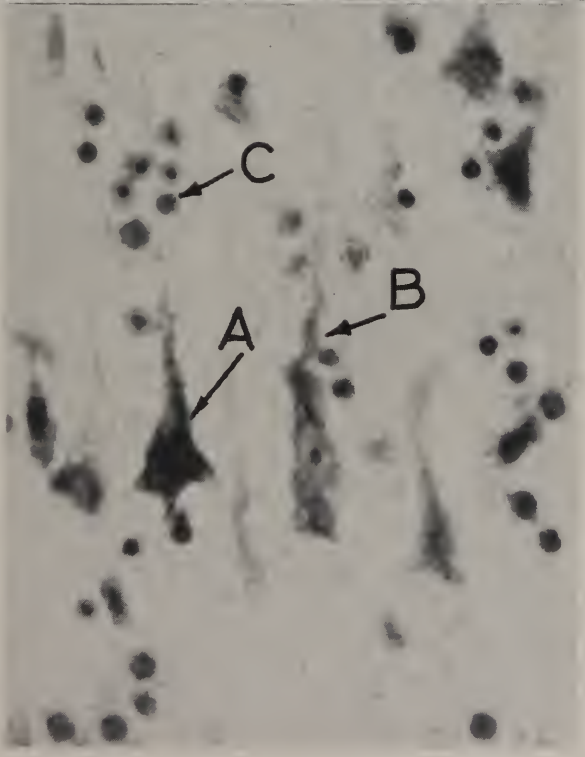
PATHOLOGY: The section is from the occipital pole. Convolutional atrophy is obvious macroscopically. In some areas the cortical lamination is fairly distinct. In others, however, the cortical pattern is considerably disarranged; especially the IIId layer shows a reduction of ganglion cells. Many of the cells that remain show evidence of degeneration: e.g., pyknosis of nuclei (A), swelling and cork-screw deformation of apical dendrites (B) and ghost-like transformation. The many small cells scattered throughout the cortex are proliferated microglia and oligodendroglia (C).

In Huntington's chorea the changes in the cortex are variable, sometimes being slight or absent.

References: Dunlap, C. B.: Pathologic changes in Huntington's chorea, Arch. Neurol. & Psychiat. 18: 867, 1927.

Wilson, S. A. K.: Neurology, Baltimore, Williams & Wilkins, 1940, p. 852.

SLIDE 40. CEREBRAL CORTEX IN HUNTINGTON'S CHOREA



NEG. 72774a X 60 NEG. 72774b X 925

HIPPOCAMPAL FORMATION IN A CASE OF EPILEPSY

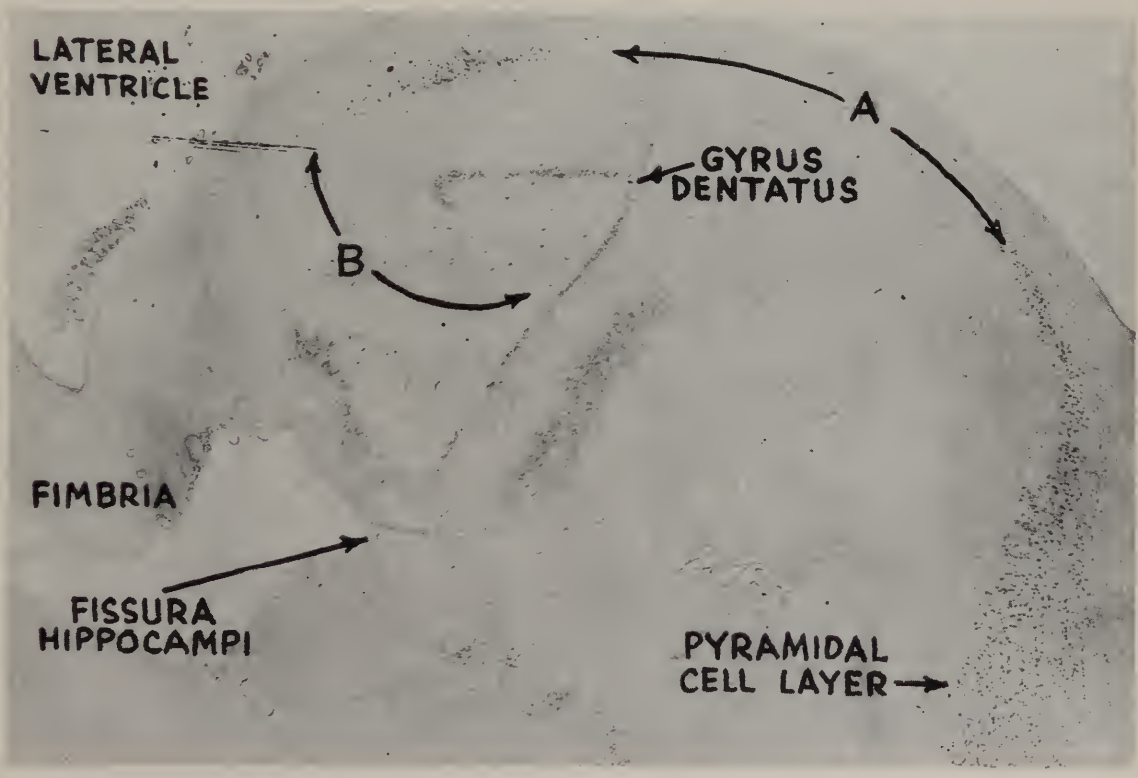
Nissl Stain

CLINICAL NOTE: Not available. (NP 83)

PATHOLOGY: There are widespread changes in the hippocampal formation. Most of the cells of the pyramidal layer are missing, particularly those of Sommer's sector (the part of the layer that bulges toward the lateral ventricle) (A) and those that approach the hilus of the gyrus dentatus (B). The granule layer in the gyrus dentatus is considerably less affected.

The cells of the pyramidal layer of the hippocampus are peculiarly prone to destruction in anoxic states, especially in epilepsy and in the toxic encephalopathies (see Slides 5, 15, 26, 41, 43 and 77).

Reference: Sommer, W.: Erkrankung des Ammonshorns als etiol. Moment der Epilepsie, Arch. f. Psychiat. 10: 631, 1880.



NEG. 72771 X 60

THE CEREBRAL CORTEX IN SCHIZOPHRENIA

Nissl Stain

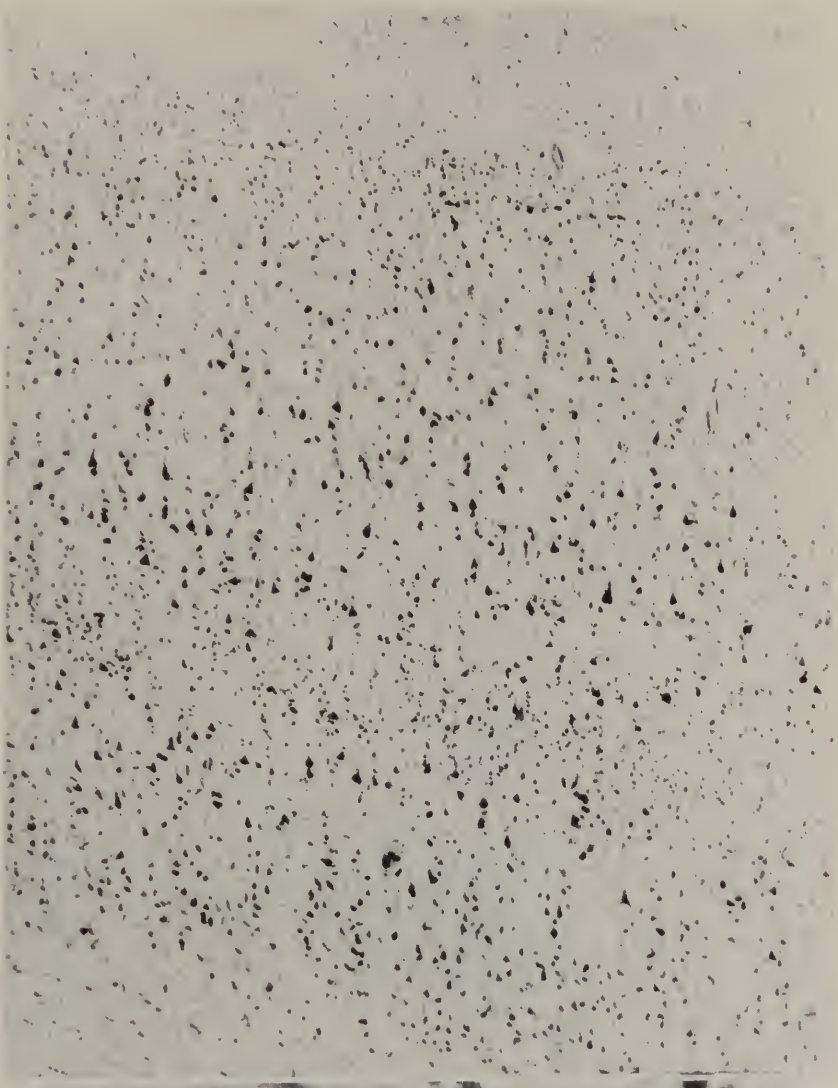
CLINICAL NOTE: A 33-year-old woman who had schizophrenia. Death was accidental. (A 3535)

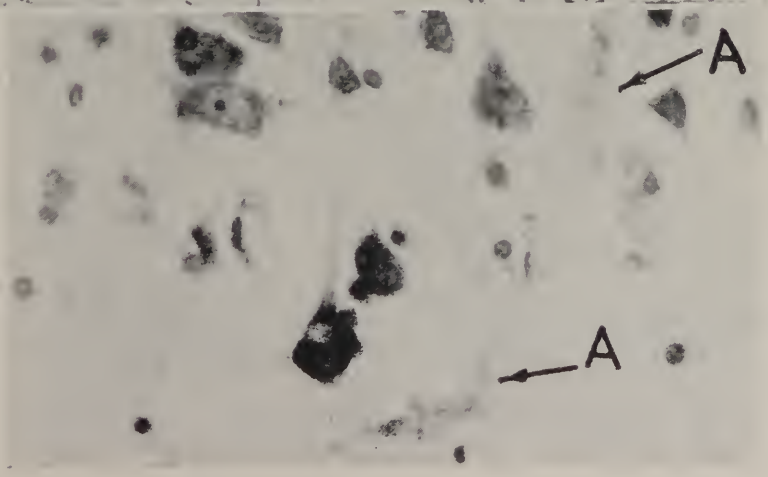
PATHOLOGY: The slide shows relatively few changes. Lamination of the cortex can be easily made out. Moderate numbers of pale-staining ganglion cells exhibiting chromatolysis are noted; others show dissolution (A). Such cells are found particularly in layer III and to a lesser extent in layers V and VI. Except for occasional satellitosis and rare neuronophagia, one sees no glial reaction.

The pathologic changes in schizophrenia vary considerably from case to case. There is no single change that can be regarded as specific.

Reference: Freeman, W.: Neuropathology. The anatomical foundation of nervous diseases, Philadelphia, Saunders, 1933, pp. 256-257.

SLIDE 42. THE CEREBRAL CORTEX IN SCHIZOPHRENIA





NEG. 72771a X 60 NEG. 72771b X 600

THE HIPPOCAMPAL FORMATION IN SCHIZOPHRENIA
(AFTER INSULIN THERAPY)
Nissl Stain

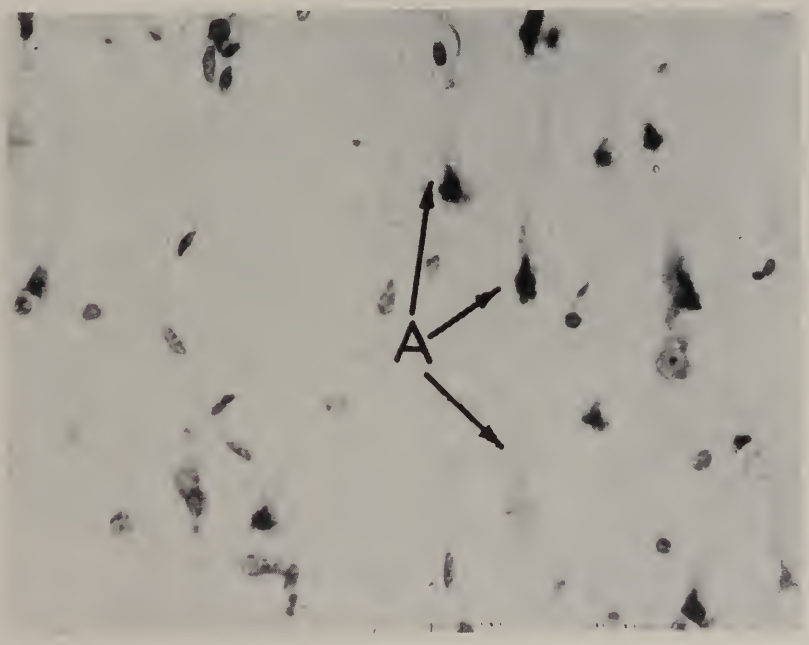
CLINICAL NOTE: The patient, a 26-year-old male with schizophrenia, died following insulin shock therapy. (NP 82)

PATHOLOGY: The section is through the hippocampal formation and adjacent temporal gyri. The cortex is similar to that described in the case of Slide 42.

In the pyramidal layer located near the transition zone between hippocampus and subiculum one notes severe and widespread destruction of the component cells. Such cells in the process of dissolution are shown in the photograph (A). There is no significant glial response in the pyramidal cell layer, but in the molecular layer (situated between the pyramidal and granular layers) considerable satellitosis and moderate neuronophagia are noted.

It seems not unlikely that the changes are due to anoxia incident to the insulin therapy.

Reference: Finley, K. H., and Brenner, C.: Histologic evidence of damage to the brain in monkeys with metrazol and insulin, Arch. Neurol. & Psychiat. 45: 403, 1941.



NEG. 72770 X 700

TUBEROUS SCLEROSIS (EPILOIA)

Holzer Stain

CLINICAL NOTE: A 23-year-old woman, born an idiot, had been epileptic since early childhood. Adenoma sebaceum was prominent. Death followed bronchopneumonia. (A 3111)

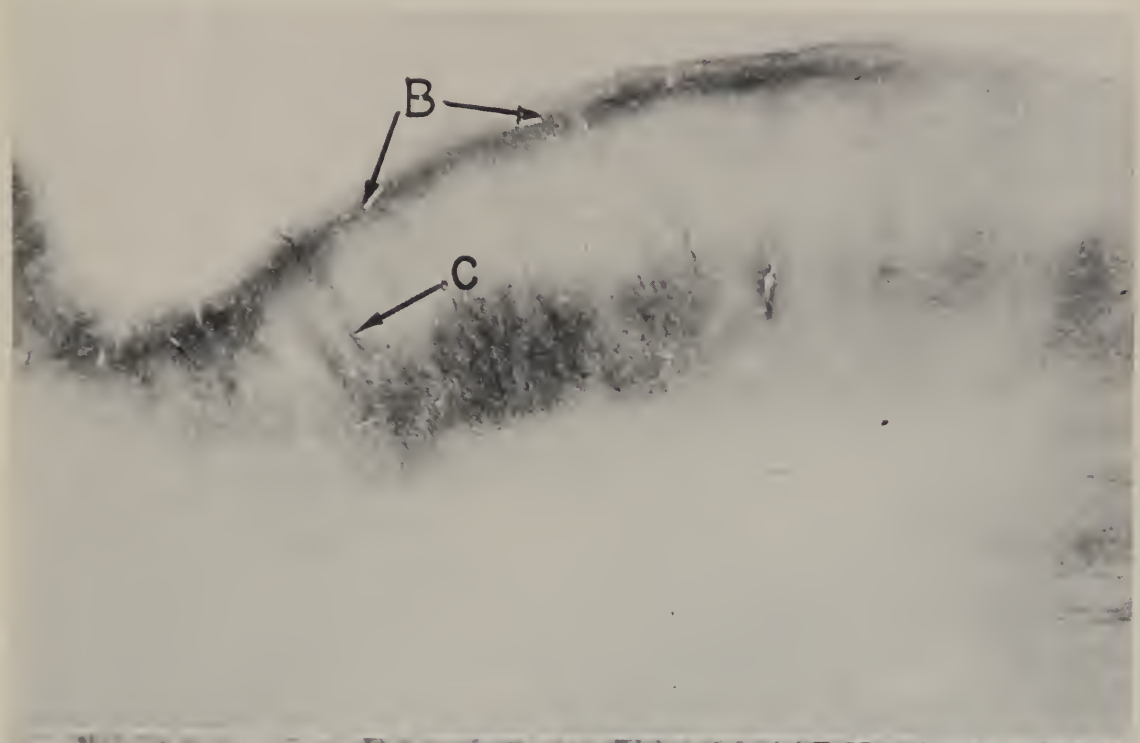
PATHOLOGY: At autopsy the kidneys were found to contain multiple small tumors (fibrolipomas). Many cerebral convolutions were broadened and hypertrophied and had the consistency of cartilage.

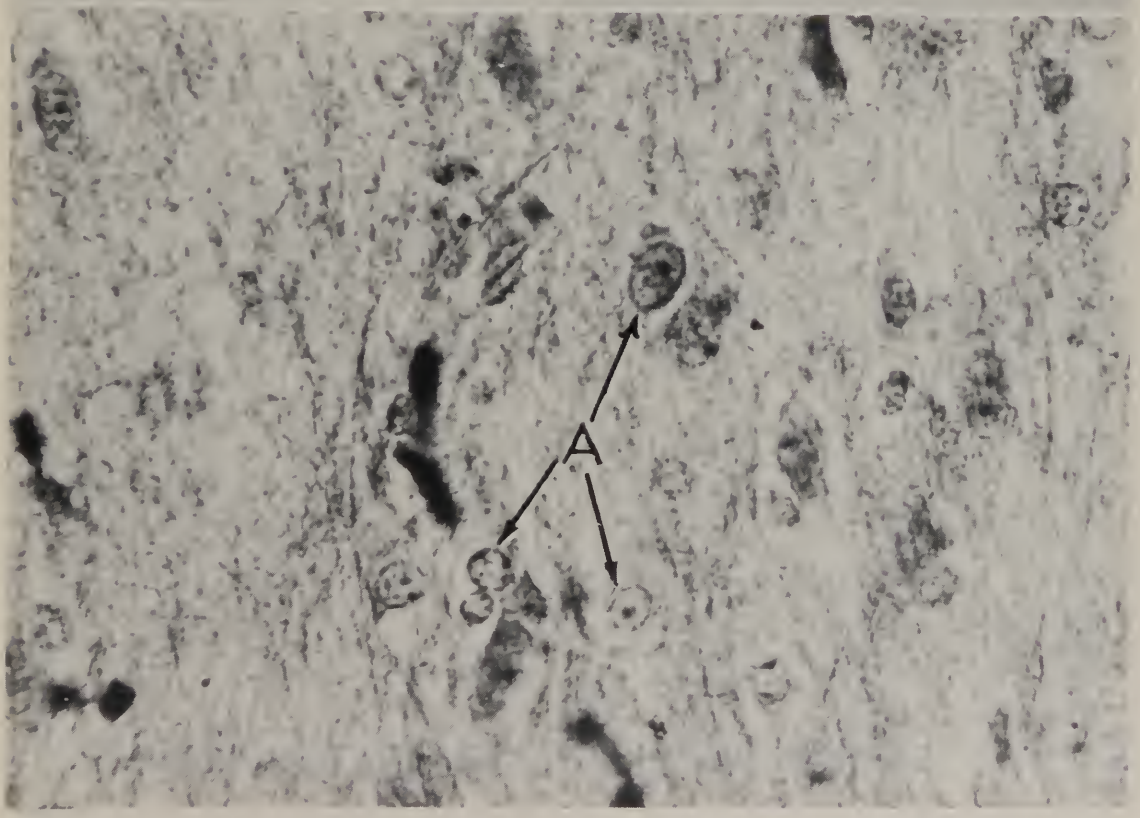
The section shows marked decrease in number of ganglion cells of the cortex. Many of those remaining are deformed. Glial cells on the other hand, are increased in number, particularly in the middle cortical layers. These cells have a vesicular nucleus (A), are unduly large so that it is difficult in some places to distinguish them from the nuclei of ganglion cells. Masses of glial fibers resembling tangled hair are most conspicuous in the outer layers of the cortex (B). In some regions the fibers form into bundles or skeins that course inward toward the white matter (C).

See also Slide 49 from same case.

Reference: Freeman, W.: Tuberous sclerosis, Arch. Neurol. & Psychiat. 8: 614, 1922.

SLIDE 44. TUBEROUS SCLEROSIS (EPILOIA)





NEG. 72782a X 15 NEG. 72782b X 915

LEUKEMIC AGGREGATES IN THE BRAIN

Hematoxylin and Eosin Stain

CLINICAL NOTE: Not available. (Case of myelogenous leukemia).

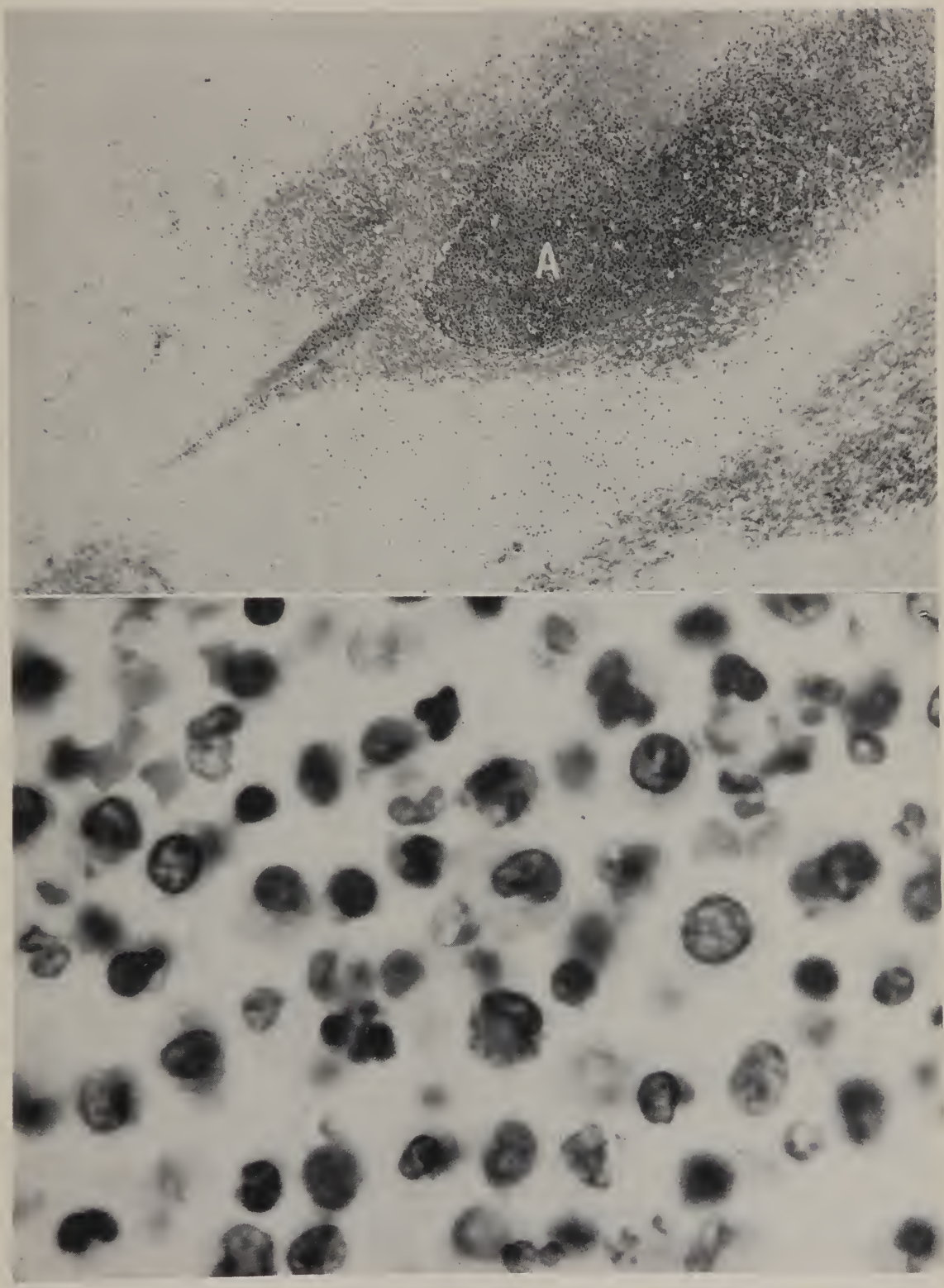
PATHOLOGY: Hemorrhages are present chiefly in the white matter. In the central portion of most of the hemorrhages there are aggregates of leukemic cells (A), the cellular detail of which is shown in the lower photograph. The blood vessels scattered throughout the section also contain leukemic cells.

In some regions where no hemorrhage has occurred one finds moderate numbers of leukemic cells in perivascular spaces. Such states may be regarded as the forerunners of leukemic infiltration of the brain. It is difficult to determine whether the hemorrhages were secondary to the establishment of such perivascular foci.

References: Schwab, R. S., and Weiss, S.: The neurologic aspect of leukemia, Am. J. Med. Sc. 189: 766, 1935.

Diamond, I. B.: Leukemic changes in the brain. A report of fourteen cases, Arch. Neurol. & Psychiat. 32: 118, 1934.

SLIDE 45. LEUKEMIC AGGREGATES IN THE BRAIN



NEG. 72995 X 100 NEG. 76850 X 1890

MICROGYRIA

Nissl Stain

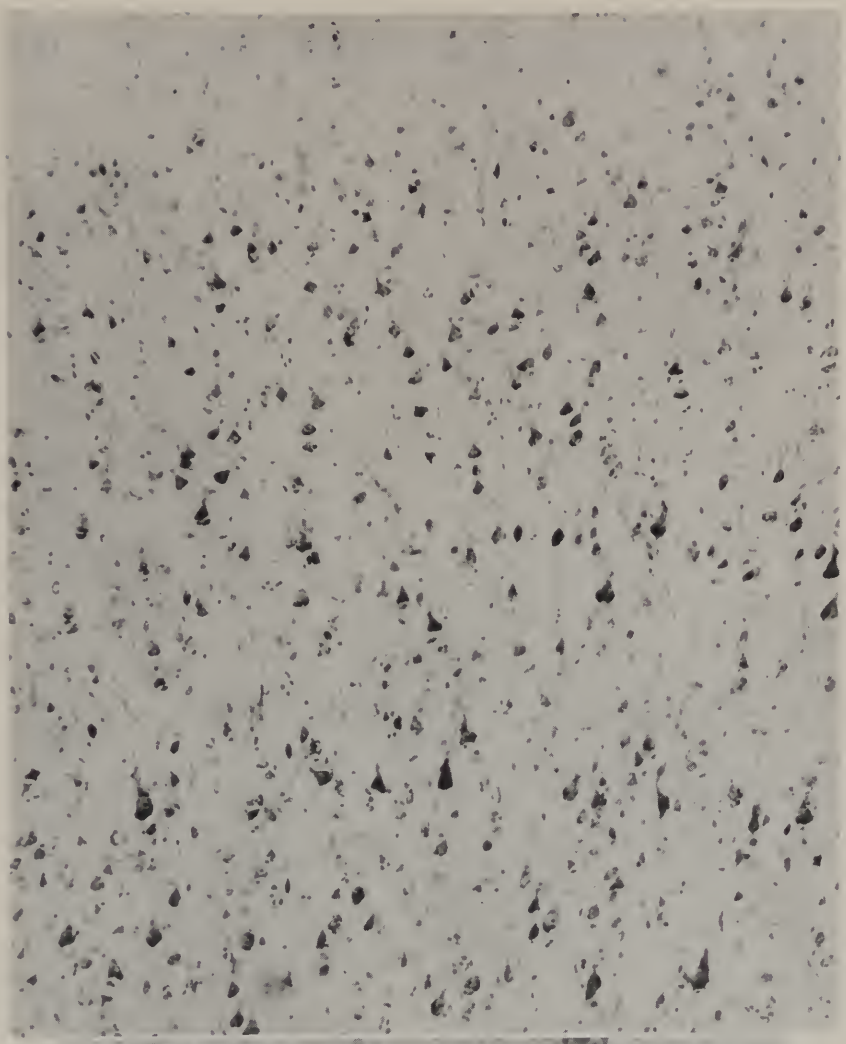
CLINICAL NOTE: Not available. (NP 156)

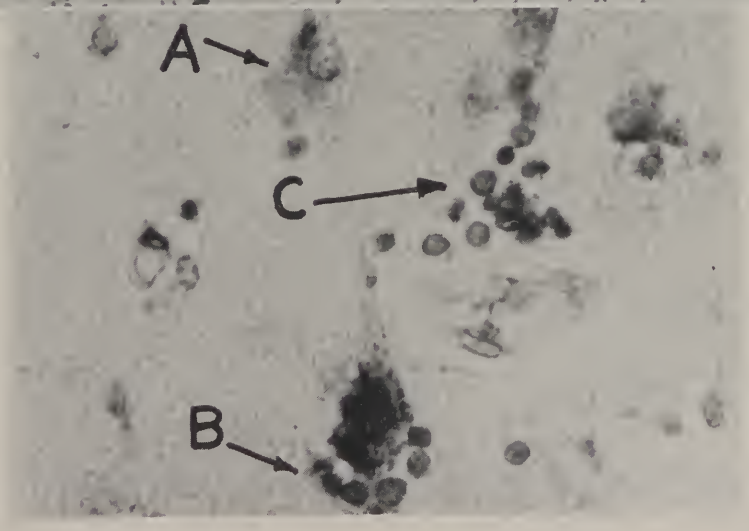
PATHOLOGY: In most of the section the lamination of the cortex is no longer discerned. In such areas the ganglion cells are widely separated and have lost all semblance of orderly arrangement; their apical dendrites no longer point toward the surface of the brain. Here and there the hyperplastic upper cortical layer dips down through the entire cortex to merge with the white matter. One also notes malformed cortical blood vessels.

Many ganglion cells are rounded or rectangular or otherwise defective. Nuclei and nucleoli are eccentric, and the cytoplasm is spongy or ghost-like (A). Neuronophagia (B) and glia-rasen ("lawns" of glia) (C) are noted most commonly in the lowest cortical layer.

Reference: Freeman, W.: Microcephaly and diffuse gliosis: A clinico-pathological study of four cases, Brain 50: 188, 1927.

SLIDE 48. MICROGYRIA





NEG. 72780a X 90 NEG. 72780b X 145

TUBEROUS SCLEROSIS (EPILOIA)

Nissl Stain

CLINICAL NOTE: Same case as in Slide 44. (A 3111)

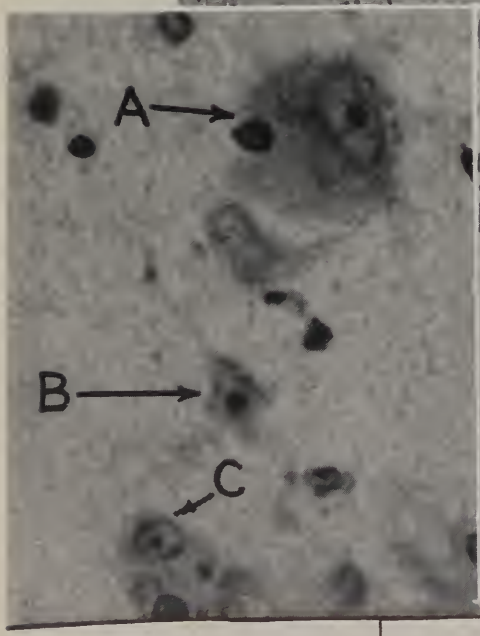
PATHOLOGY: The cortex is laminated but in "gross" architecture is defective owing to the presence of linear columns devoid of ganglion cells, which extend through the cortex into the white matter. The transition between cortex and white matter is indistinct since ganglion cells of the lower cortical layer are strewn throughout the adjacent white matter. In the lowest cortical layer one notes that many of the cells have lost their orientation with respect to the cortex, their processes tending to be unduly parallel or tangential to the surface of the brain rather than perpendicular.

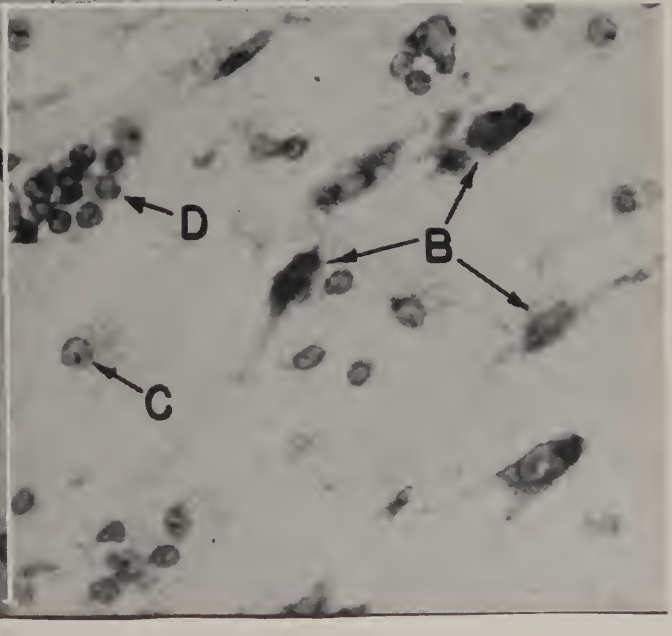
The ganglion cells vary considerably in size and many have taken on bizarre shapes. Some of the latter have ballooned cytoplasm and eccentric nucleus (A) while others are small and shrunken (B). Here and there one sees giant ganglion cells with fenestrated and vacuolated cytoplasm.

Rod cells (reactive microglia) permeate the cortex. Here, astrocytes (C) are also to be found in abundance. The oligodendroglial satellites too are proliferated (satellitosis). In the lower layer of the cortex one notes glia-rasen, or "lawns" of glia, which have replaced destroyed ganglion cells: these are mostly oligodendroglia (D).

Reference: Critchley, M., and Earl, C. J. C.: Tuberous sclerosis and allied conditions, Brain 53: 311, 1932.

SLIDE 49. TUBEROUS SCLEROSIS (EPILOIA)





NEG. 72778a X 40 | NEG. 73834 X 950 NEG. 72778b X 705

SUBACUTE COMBINED DEGENERATION OF THE SPINAL CORD

Fat Stain

CLINICAL NOTE: A 61-year-old female with pernicious anemia whose signs and symptoms indicated marked involvement of the pyramidal tracts and the posterior columns of the spinal cord. (A 3483)

PATHOLOGY: In the pyramidal tracts there is a comparatively recent demyelination, imparting to the areas involved a honeycombed or cribiform appearance (A). Within this focus one notes large gitter cells containing the fatty debris of disintegrated myelin.

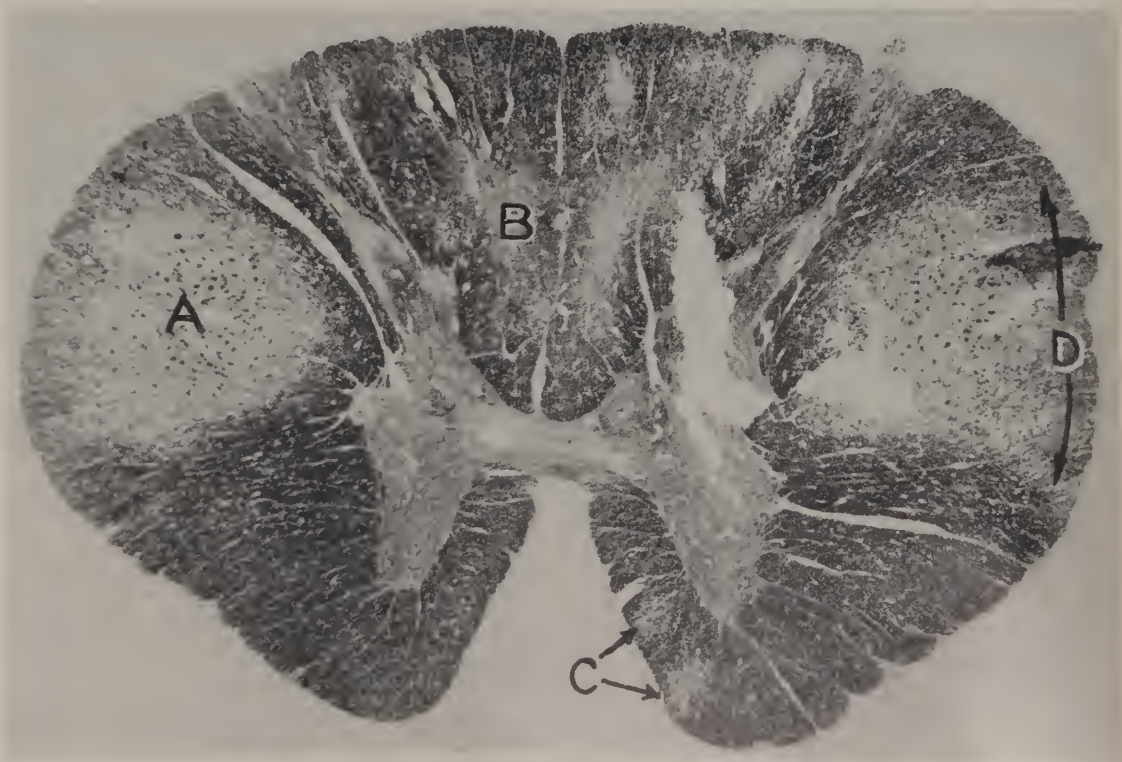
In the posterior columns an old focus of demyelination is to be seen (B). Within perivascular spaces there are a few fat-laden gitter cells which are doubtless en route to enter the circulation.

Small old foci are present also in the region of the anterior column (C) and in the spinocerebellar tracts (D).

In the grey matter the faint shadows of ganglion cells can be made out. Although a few appear to have undergone fatty degeneration, most show no deviation from the normal.

(See also Slide 73 in which the posterior columns are more affected.)

Reference: Wilson, S. A. K.: Neurology, Baltimore, Wm. Wood, 1940, vol. 2, pp. 1347-1349.



Slide 50 NEG. 72779 X 15

TABES DORSALIS

Loyez Myelin Sheath Stain

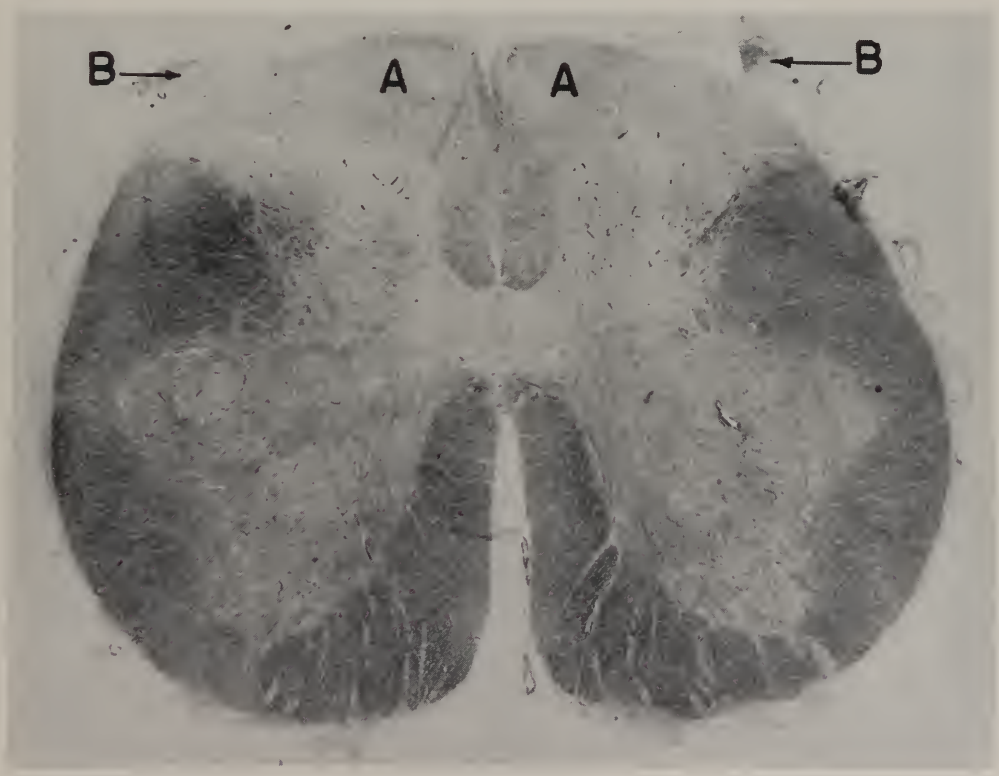
CLINICAL NOTE: Not available.

PATHOLOGY: The section is from the lower lumbar region of the spinal cord. Demyelination of the posterior columns (A) is conspicuous. Some of the myelin sheaths in the more medial parts of the posterior column are relatively intact, indicating that fibers entering from lower sacral segments are not implicated. Many of the myelin sheaths that remain are fragmented, swollen and otherwise distorted.

The ganglion cells of the posterior horns are intact as judged both by their microscopic appearance and by the integrity of myelinated fibers in the lateral and anterior columns.

The stubs of posterior roots (B) are visible in the section. Virtually all the fibers are devoid of myelin.

Reference: Stern, R. O.: A study of histopathology of tabes dorsalis, with special reference to Richter's theory of its pathogenesis, Brain 52: 295, 1929.



Slide 51 NEG. 76613 X 15

AMAUROTIC FAMILY IDIOCY

Nissl Stain

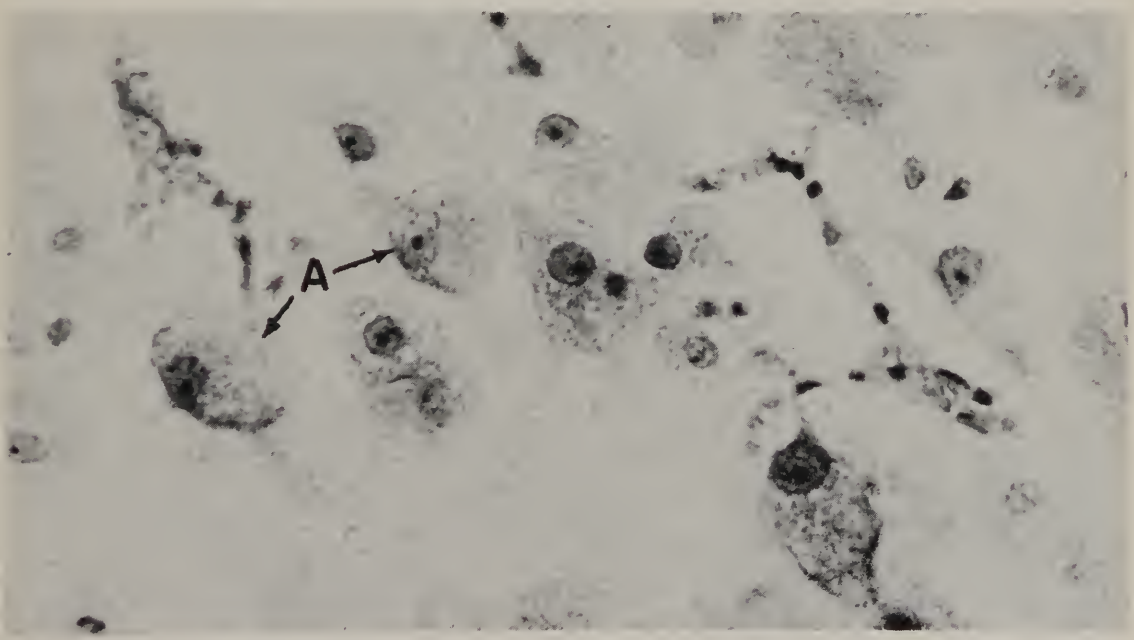
CLINICAL NOTE: Not available. (NP 157)

PATHOLOGY: This is the typical picture of amaurotic family idiocy. Lamination in the cortex is difficult to make out. The ganglion cells are considerably swollen and have taken on globular, pear- or bottle-shaped forms (A). The nuclei are eccentric and contain a well preserved nuceolus.

The cytoplasm is pale and somewhat vacuolated. In many cells the cytoplasm possesses a fine granular network. Prelipids are thought to be contained in the cytoplasm; these are not visible in Nissl stains but take on a pale orange hue with scarlet red.

Virtually no normal ganglion cells are to be found. Throughout the section there are many gitter cells with markedly honeycombed or vacuolated cytoplasm.

Reference: Hassin, G. B.: Amaurotic family idiocy, clinical and pathologic studies, Am. J. Psychiat. 8: 969, 1929.



NEG. 72823 X 810

Slide 52

DELAYED TRAUMATIC APOPLEXY

Nissl Stain

CLINICAL NOTE: Same case as in Slides 8 and 60. (A 3704)

PATHOLOGY: Two features mark the section. On the one hand there is evidence of an old injury of the cortex, as manifested by disappearance of many cortical ganglion cells, neuronophagia, an advanced proliferation of vessels (A), myriads of rod cells (B) and many astrocytes (C).

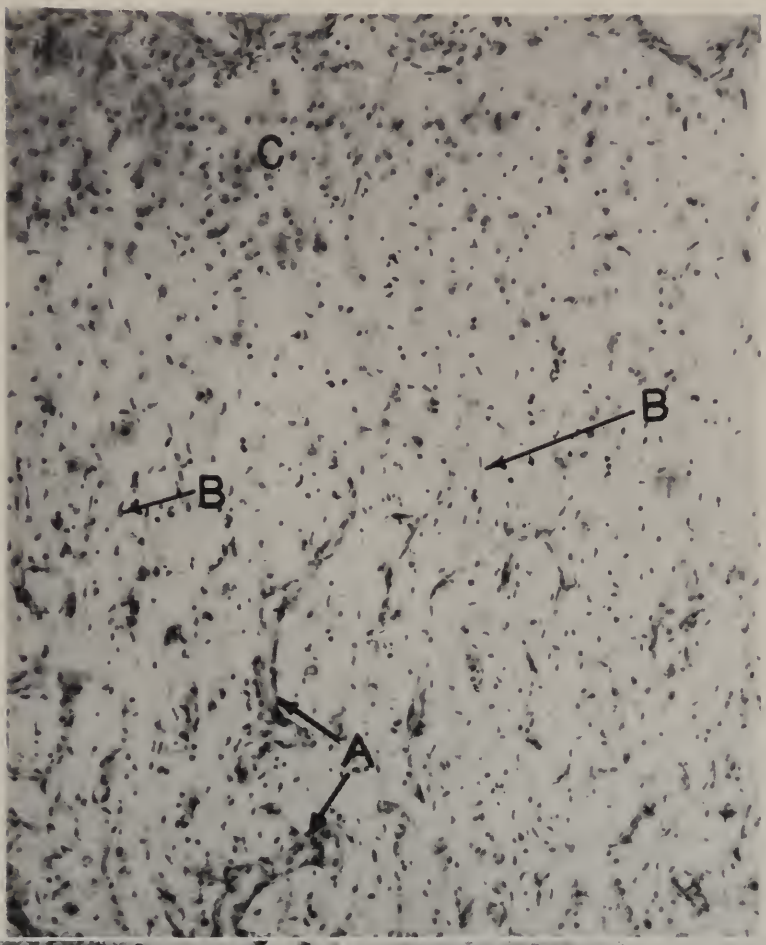
On the other hand, hemorrhages of fairly recent origin are to be found in both leptomeninges and the cortex and to a lesser degree in the subcortical white matter (D). Some of the hemorrhages are immediately perivascular (E).

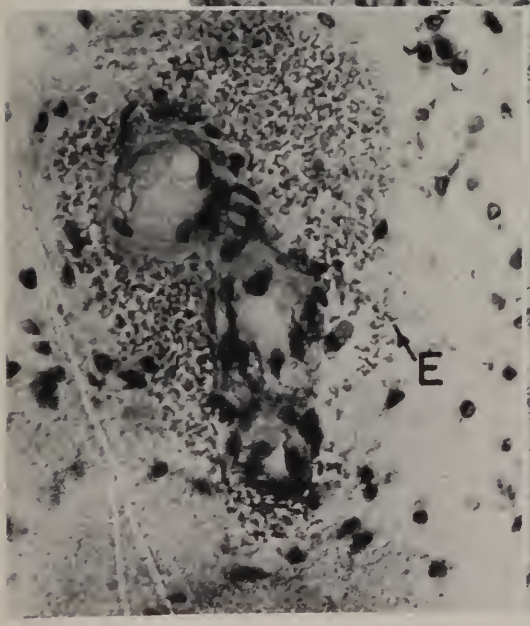
It is concluded that the hemorrhages have been secondarily induced at the sites of old cortical scarring.

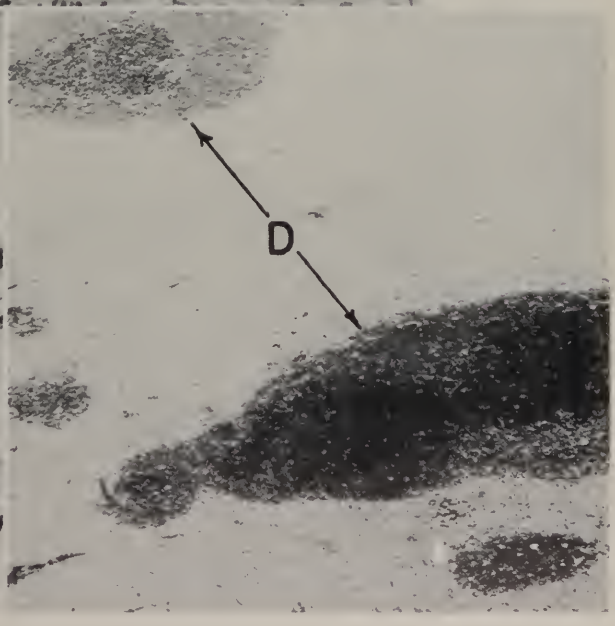
References: Bollinger, O.: Ueber traumatische Spat-Apoplexie. Ein Beitrag zur Lehre von der Hirnschutterung, Intl. Beit. z. Wissenschaftlichen Med., Festschr. Rudolph Virchow, Berlin, vol. 2, 1891.

Courville, C. B., and Blomquist, O. A.: Traumatic intracerebral hemorrhage, with particular reference to its pathogenesis and its relation to "delayed traumatic apoplexy", Arch. Surg. 41: 1, 1940.

SLIDE 53. DELAYED TRAUMATIC APOPLEXY







NEG. 74153 X 100 NEG. 73891 X 400 NEG. 72768 X 60

CHRONIC SUBDURAL HEMATOMA

Hematoxylin and Eosin Stain

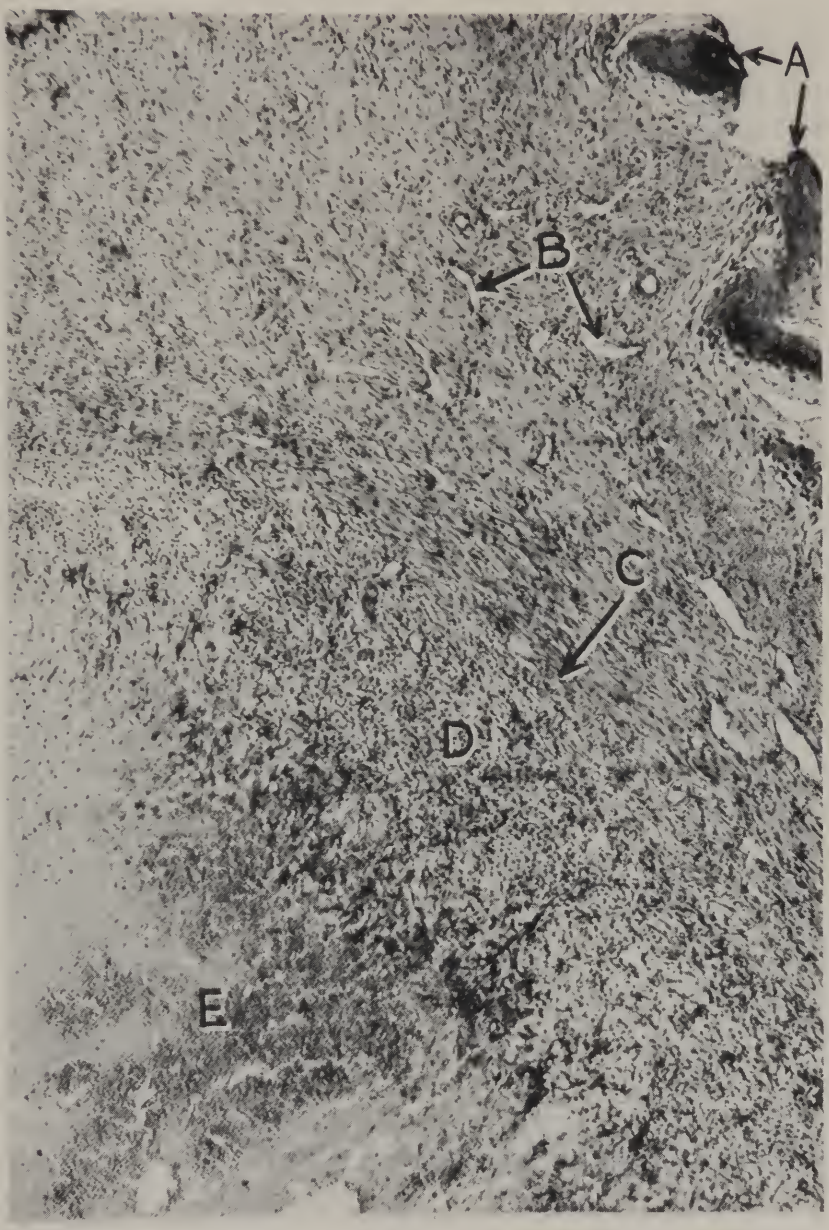
CLINICAL NOTE: A male, aged 13, who developed headache and diplopia following traumatic injury of the head. The cerebrospinal fluid pressure ranged between 200 and 550 mm. Hg. Exploratory craniotomy revealed an organized subdural hematoma overlaying the left parietal and temporal lobes. (NP 109)

PATHOLOGY: The section is composed of dura as well as a subdural hematoma. The outer part of the dura contains spicules of bone (A) and is greatly distended with hemorrhage. Scattered throughout the dura are characteristic empty spaces (B). The approximate line of cleavage between dura and the outer layer of the hematoma is indicated by C.

The outer layer of the hematoma is composed of granulation tissue (D): Macrophages containing blood pigment are prominent. Numerous fibroblasts invade the periphery of the clot. The blood clot (E) is not organized. The inner aspect of the hematoma is devoid of mesothelium except in an area or two where a thin single-celled layer can be made out.

Reference: Putnam, T. J., and Cushing H.: Chronic subdural haematoma. Its pathology, its relation to pachymeningitis haemorrhagica and its surgical treatment, Arch. Surg. 11: 329, 1925.

SLIDE 57. CHRONIC SUBDURAL HEMATOMA



NEG. 72775 X 75

PACHYMENINGITIS HEMORRHAGIC INTERNA

Hematoxylin and Eosin Stain

CLINICAL NOTE: A male, aged 71, was admitted to hospital unconscious. He was found to have a left-sided hemiplegia. Two-and-one-half weeks prior to admission he suddenly developed severe headache. There had been no history of trauma. His blood pressure on admission was 160 systolic, 100 diastolic. Death occurred the day after admission. (A 3453)

PATHOLOGY: Autopsy revealed generalized arteriosclerosis, pneumonia and right-sided subdural hemorrhage which considerably compressed the brain.

The section shows a greatly thickened dura containing many endothelium-lined channels (A) beneath which is an organizing hematoma. Some of the channels contain blood.

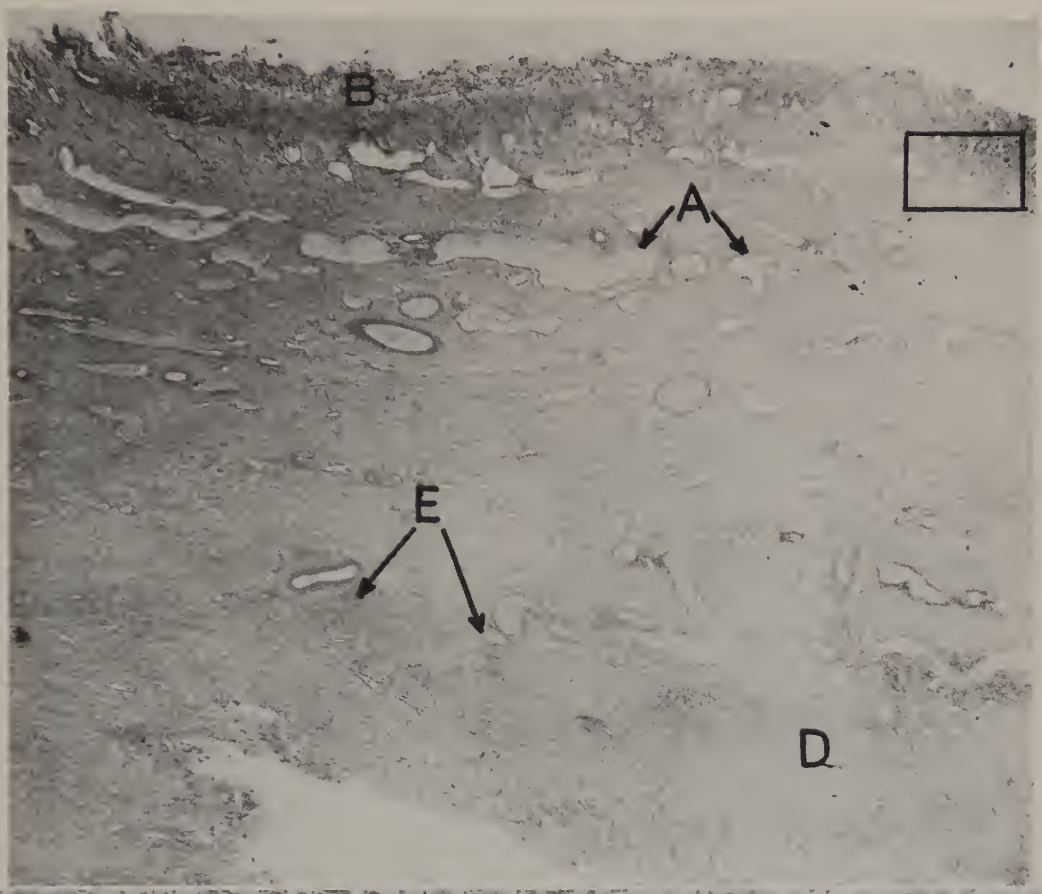
In the outermost zone of the dura one notes a rather thin layer of granulation tissue (B) containing small round cell accumulations and a few histiocytes and giant cells (C).

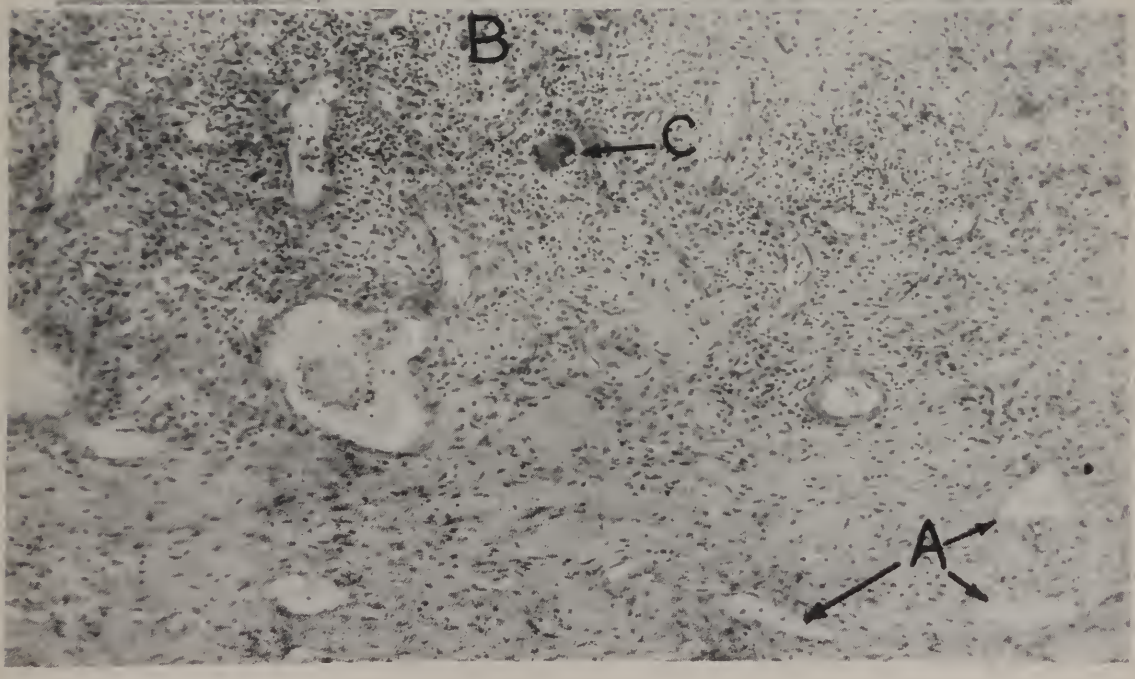
The inner boundary of the dura cannot be made out because of the granulation tissue (D) with which it is contiguous. The approximate line of cleavage of dura and granulation tissue (inner layer of the subdural hematoma) is indicated by E in the photograph. The subdural hematoma is similar to that described in Slide 57.

The essential difference in the two slides is that in Slide 58 there is abundant inflammatory reaction and hemorrhage within the dura, especially its inner portion, hence the term, pachymeningitis hemorrhagica interna.

Reference: Griswold, R. A., and Jelsma, F.: The relationship of chronic subdural haematoma and pachymeningitis haemorrhagica interna, Arch. Surg. 15: 45, 1927.

SLIDE 58. PACHYMENINGITIS HEMORRHAGICA INTERNA





NEG. 74049 X 26 NEG. 72822 X 125

MENINGITIS, PROBABLY BLASTOMYCOTIC
Hematoxylin and Eosin Stain

CLINICAL NOTE: A 25-year-old woman who had signs and symptoms indicative of chronic meningitis. A clinical diagnosis of syphilitic meningitis had been made. (NP 73)

PATHOLOGY: The section shows a greatly thickened pia-arachnoid composed of granulation tissue with superimposed fibrino-purulent exudate. Within the granulation tissue (A), and not infrequently within multinucleate giant cells (B), there are spherical double-contoured bodies surrounded by a fairly wide capsule. No buds or mycelia are visible.

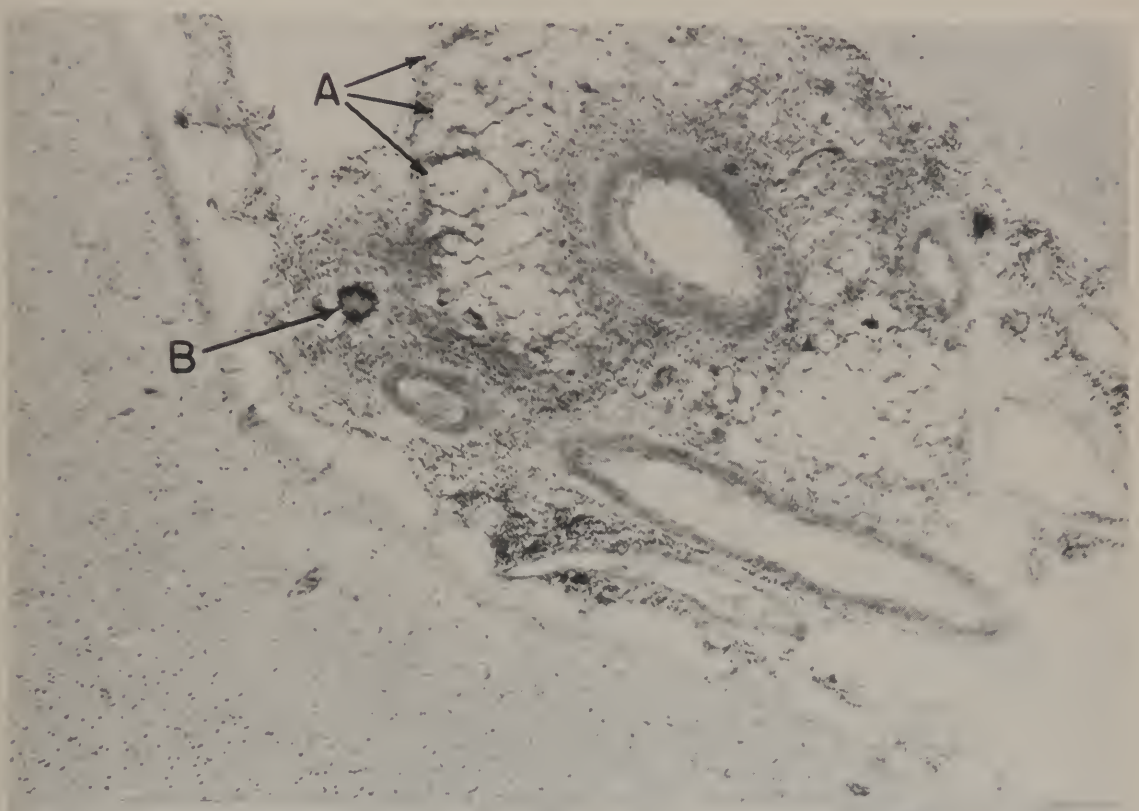
In some of the sections the bodies form large aggregates while in others the bodies are thinly scattered.

The abundance of organisms and the lysis in the fibrino-purulent exudate suggests torula histolytica infection but the size and double-contoured capsule favors the diagnosis of blastomycosis.

References: Wilhelmj, C. M.: The primary meningeal form of systemic blastomycosis, Am. J. Med. Sc. 169: 712, 1925.

Freeman, W.: Torula infection of the central nervous system, J. f. Psychol. u. Neurol. 43: 236, 1931.

SLIDE 59. MENINGITIS, PROBABLY BLASTOMYCOTIC



NEG. 72827 X 75 NEG. 76623 X 1100

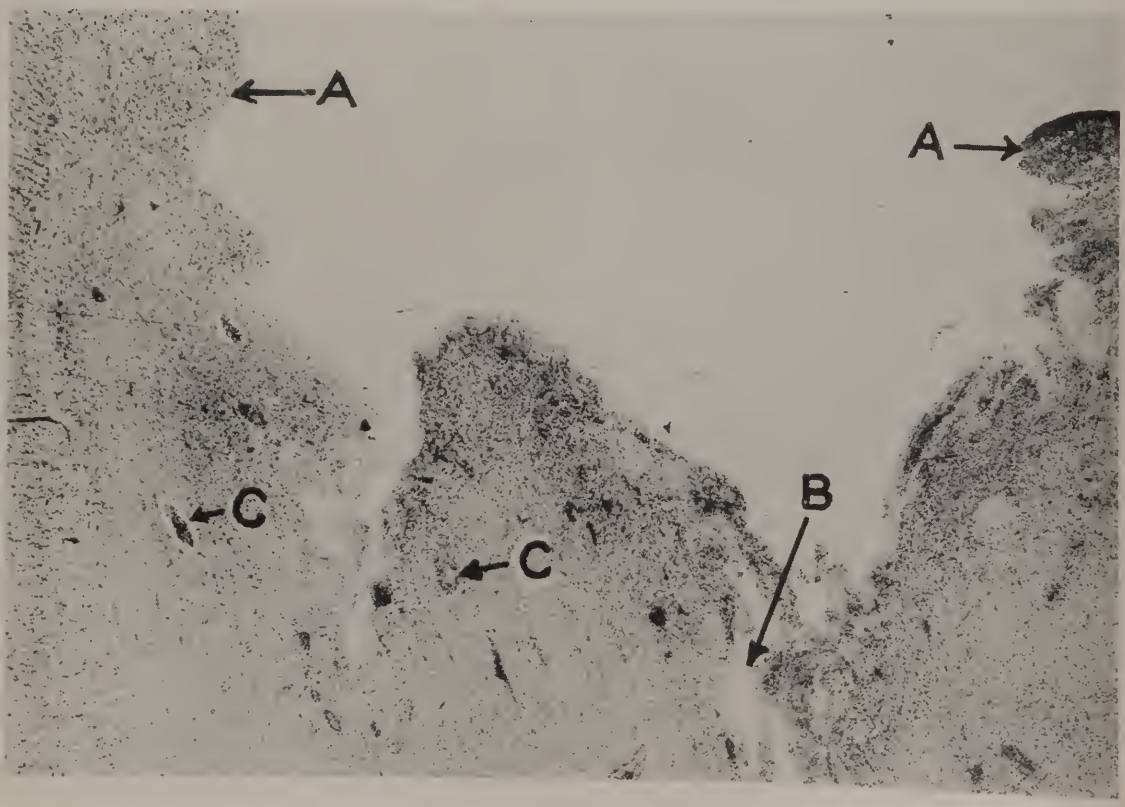
OLD TRAUMATIC CEREBRAL SCAR

Nissl Stain

CLINICAL NOTE: Same case as in Slides 8 and 53. (A 3704)

PATHOLOGY: A crater-like depression marks the region of the scar. The edge of the cortex is indicated in the photograph by A. The cortex of the brain has virtually disappeared. The scarred area itself is fissured (B). In the meshes of the perivascular spaces there are dense accumulations of gitter cells (compound granular corpuscles) (C). Throughout the tissue one notes many swollen astrocytes, gitter cells, and oligodendroglia cells.

Reference: Hassin, G. B.: General pathological considerations in brain injury. In Brock, S., Injuries of the skull, brain and spinal cord, Baltimore, Williams & Wilkins, 1940.



NEG. 74233 X 10

SYRINGOMYELIA

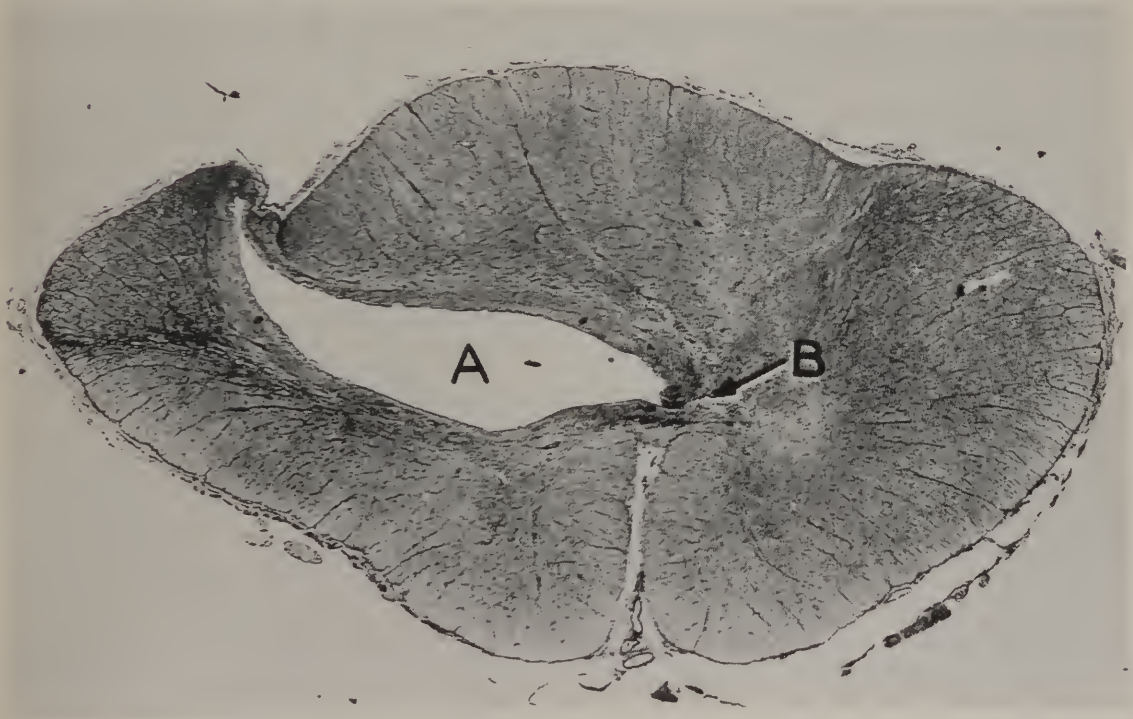
Van Gieson Stain

CLINICAL NOTE: Not available. (NP 165)

PATHOLOGY: The syringomyelic cavity (A) extends from the vicinity of the central canal (B) to the region of entrance of the dorsal root. Much of the dorsal horn on that side has been destroyed.

The lumen of the cavity is lined by wavy, sparsely cellular fibrillar connective tissue. Deeper in the wall one meets proliferated glia.

Reference: Tauber, E. S., and Langworthy, R. R.: Syringomyelia and formation of cavities in the spinal cord, Nerv. & Mental Dis. 81: 245, 1935.



NEG. 72832 X 15

Slide 62

INTRAMEDULLARY GLIOBLASTOMA MULTIFORME

Hematoxylin and Eosin Stain

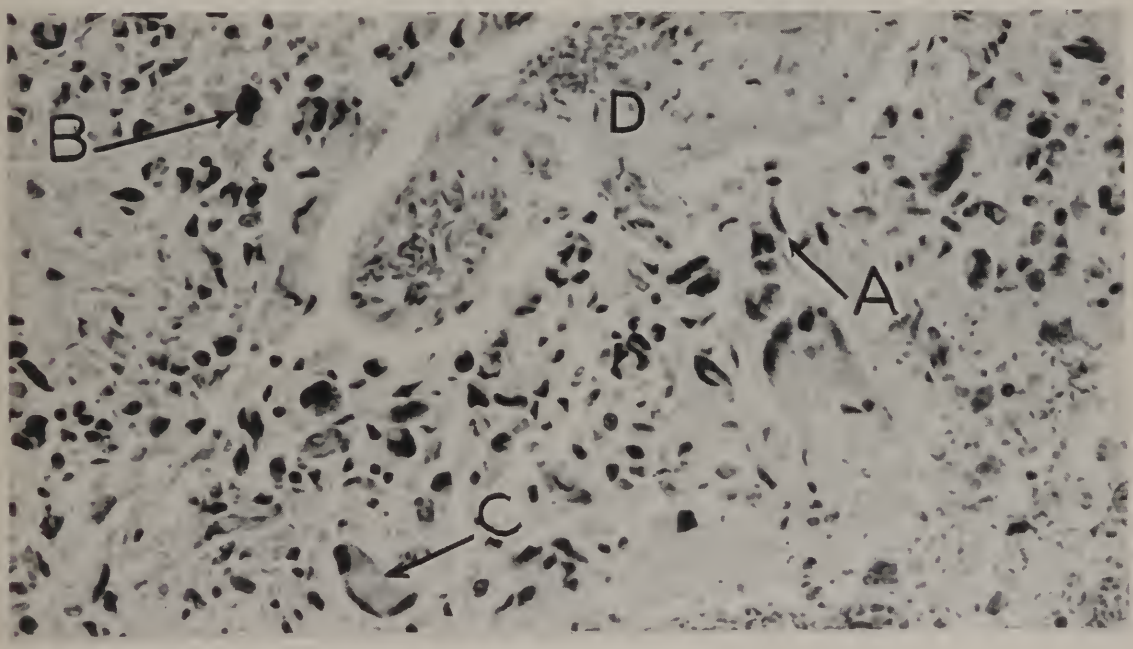
CLINICAL NOTE: Not available. (NP 1)

PATHOLOGY: Most of the substance of the cord is replaced by tumor tissue. The neoplastic cells vary greatly in size and appearance. Some of the smaller ones have the appearance of spongioblasts (A). There are numerous giant cells, some mononuclear (B), others polynuclear (C). Mitoses are common.

The tumor is well vascularized. The walls of some of the vessels are in a state of degeneration (D), or of proliferation, which probably accounts for the hemorrhages and focal necroses.

Glioblastoma multiforme is an uncommon spinal cord tumor; the most frequently occurring type is the ependymoma.

Reference: Elsberg, C. A.: Tumors of the spinal cord, Arch. Neurol. & Psychiat. 22: 949, 1929.



NEG. 72825 X 225

Slide 63

GLIOBLASTOMA MULTIFORME OF THE BRAIN

Hematoxylin and Eosin Stain

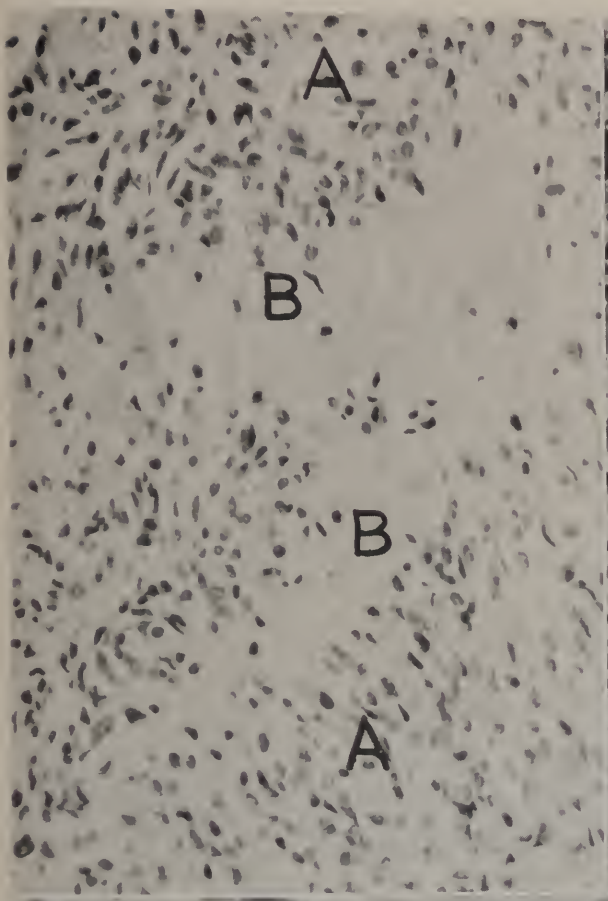
CLINICAL NOTE: A male, 47 years old, who over a period of a few months developed headache, and paresis of the left side. On admission he was comatose. Operation disclosed an extensive hemorrhagic tumor in the right parietal region. Only a few fragments were removed. (SP 31472)

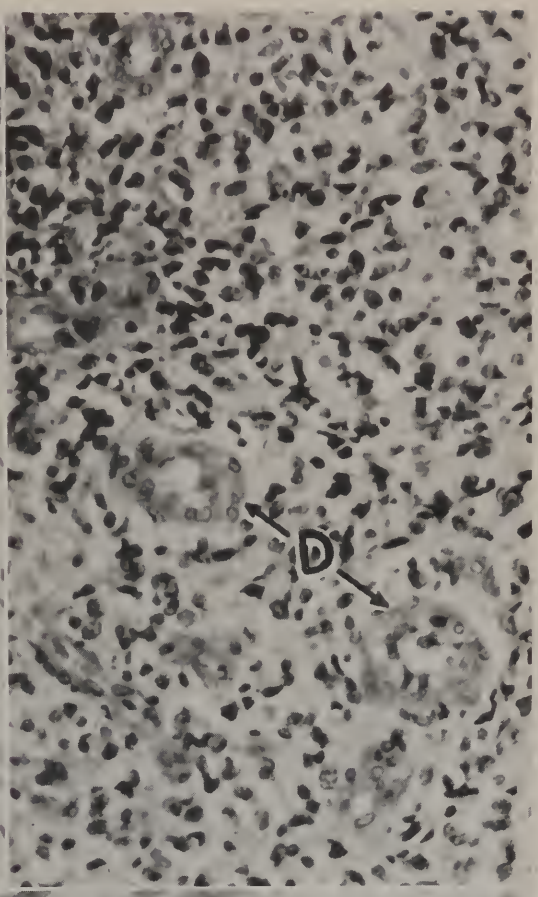
PATHOLOGY: The section shows features somewhat similar to those in Slide 63. The most common cell type conforms to the spongioblast, the cells being fusiform with comparatively small nuclei and fairly abundant cytoplasm. In places these cells form pseudo-palisades (A) where they border spongy necrotic areas (B). A sprinkling of bizarre multinucleate cells is noted (C). The vessels are increased in number and show proliferation of their walls (D) as well as degenerative changes.

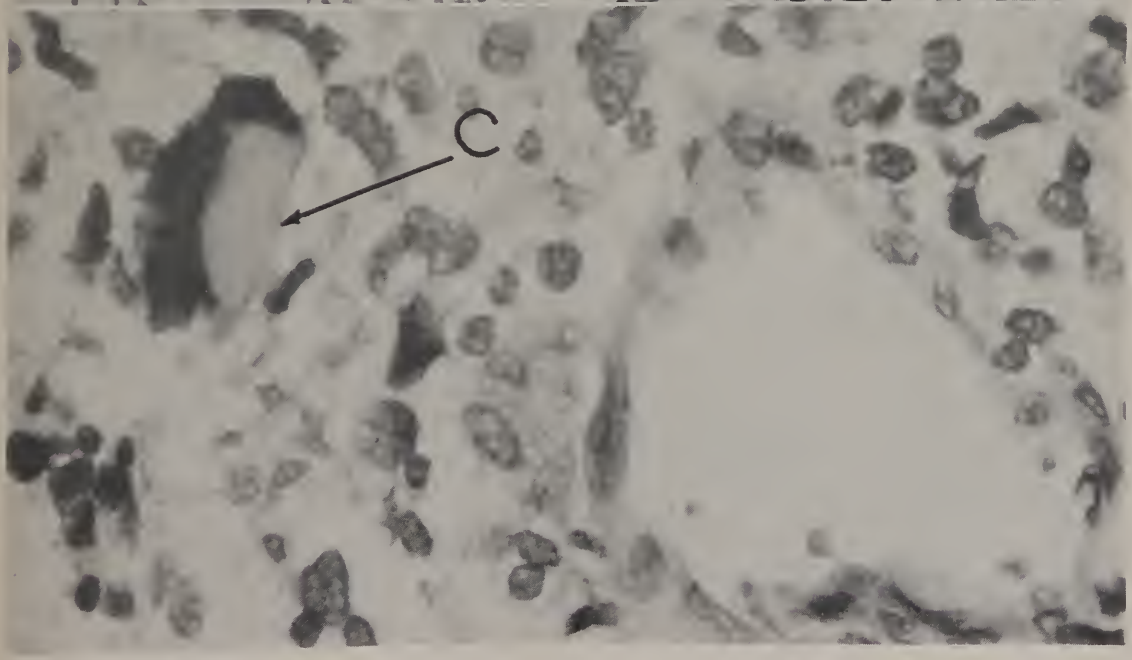
This is the most frequently occurring intracranial tumor.

Reference: Penfield, W.: A paper on classification of brain tumors and its practical application, Brit. M. J. 1: 337, 1931.

SLIDE 64. GLIOBLASTOMA MULTIFORME OF THE BRAIN







NEG. 72854 X 250 NEG. 72829 X 810

CHROMOPHOBE ADENOMA OF THE HYPOPHYSIS

Hematoxylin and Eosin Stain

CLINICAL NOTE: Not available.

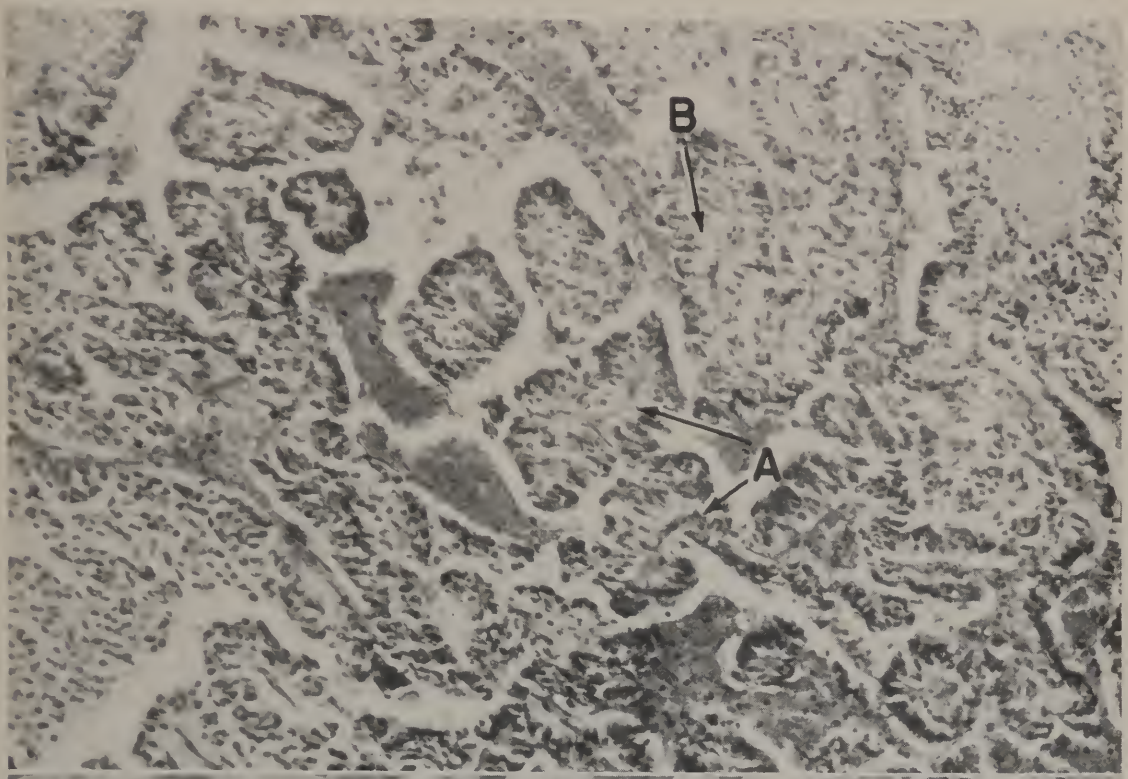
PATHOLOGY: The adenoma occupies a relatively large part of the anterior lobe of the hypophysis. It is permeated by various sized vascular channels lined by a single layer of cells, which is a characteristic feature of these tumors. The stroma is scanty.

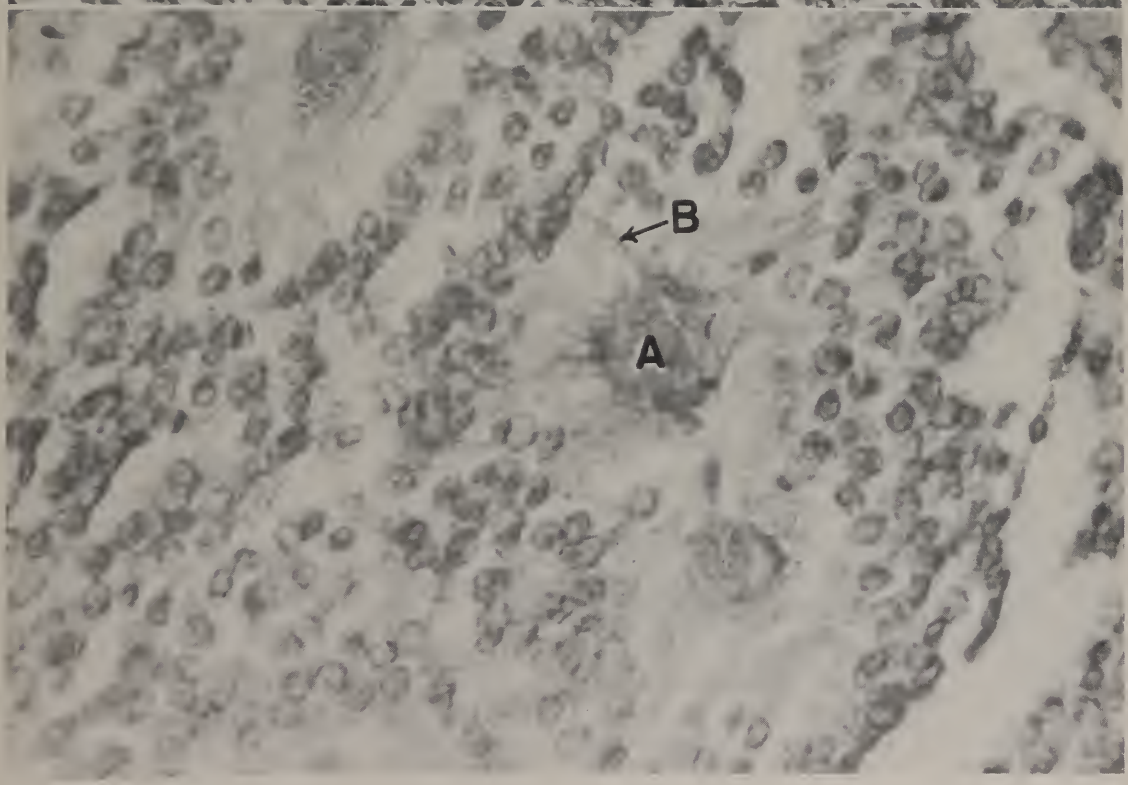
The tumor cells have taken on a pseudo-alveolar pattern. Individual pseudo-alveoli are composed of several layers of cells surrounding a central core of fibrillary material which frequently contains a capillary (A). In places the "fibrillary" material resolves itself into clear cut cytoplasmic projections attached to the central vessel (B). Here and there the fibrillary core has undergone collagenous change.

The cells contain an oval or round vesicular nucleus. The cytoplasm of most of the cells is fenestrated and its borders poorly defined. Frequently the cytoplasm is continuous into long polar processes; on occasion the cells are polygonal. In some cells, arranged mostly in clumps, the cytoplasm is faintly granular.

Reference: Dott, N. M., and Bailey P.: Hypophysial adenomata, Brit. J. Surg. 13: 314, 1925.

SLIDE 67. CHROMOPHOBE ADENOMA OF THE HYPOPHYSIS





NEG. 76614 X 200 NEG. 76858 X 640

PERIPHERAL NEURITIS

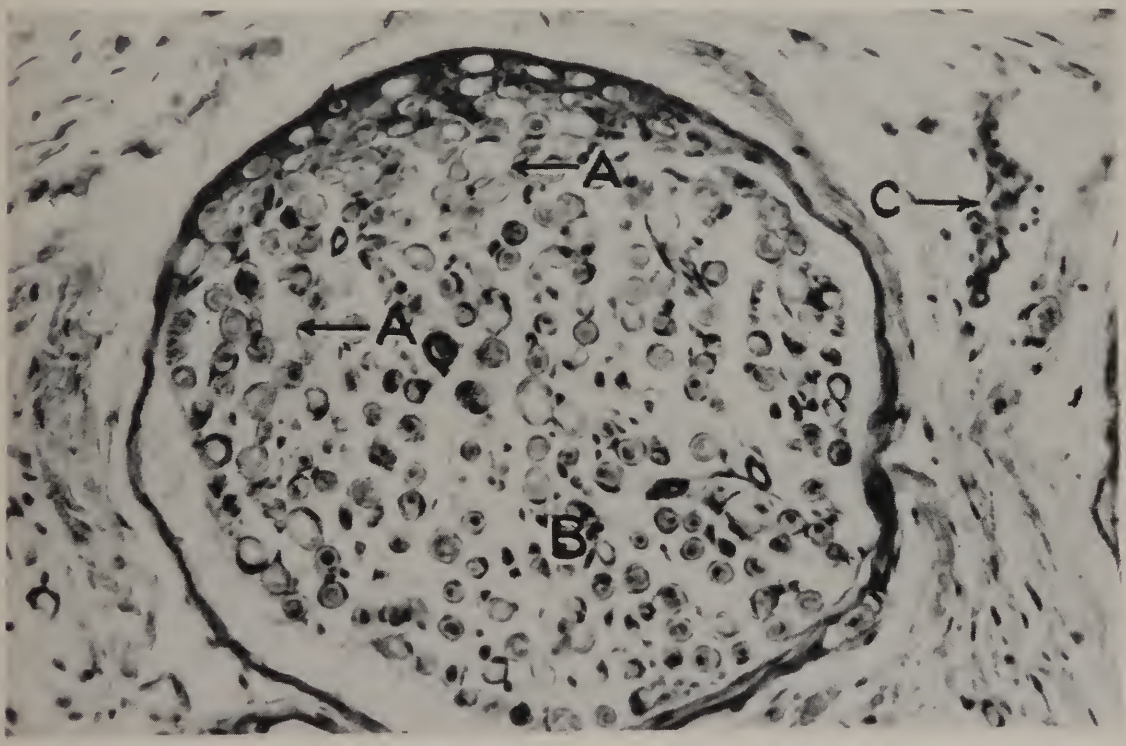
Hematoxylin and Eosin Stain

CLINICAL NOTE: A 45-year-old male with severe sciatica who committed suicide. (MP 3036)

PATHOLOGY: This is a cross section through the 5th lumbar nerve. There is degeneration of myelinated fibers, some of which appear as empty rings (A). One gains the impression that a moderate number of fibers have dropped out.

A slight cellular infiltrate is seen in the endoneurium of the bundle photographed (B). In an adjoining bundle not visible in the photograph, the infiltrate, consisting of small round cells, is abundant, and there appears to be an increase of endoneurial tissue. In the epineurium, one notes small collections of round cells in perivascular spaces (C).

Reference: Hassin, G. B.: Peripheral nerves; anatomic and pathologic considerations, Arch. Neurol. & Psychiat. 27: 57, 1932.



NEG. 72855 X 300

CRANIOPHARYNGIOMA

Hematoxylin and Eosin Stain

CLINICAL NOTE: A boy, aged 8, had had over a period of years attacks of headache and vomiting. Occasionally there were convulsive seizures. More recently his vision failed and a moderate hydrocephalus developed. At operation a partially cystic tumor was removed from the region of the base of the brain adjacent to the pituitary gland. (SP 22202)

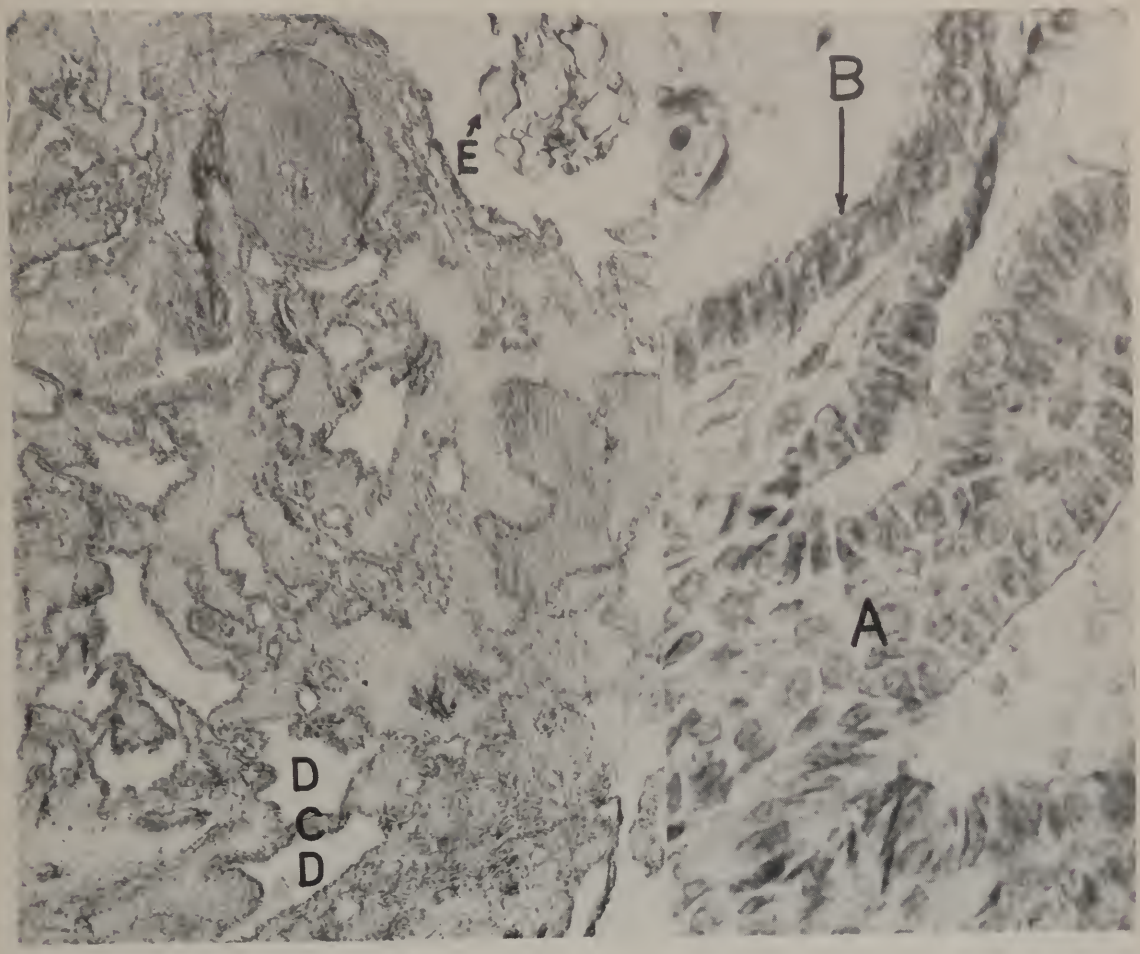
PATHOLOGY: The picture is that of a squamous epithelial papillary tumor. There are masses of epithelial cells (A) surmounted by a columnar basal layer (B). Interweaving epithelial columns or offshoots (C) bridge the many small cyst-like spaces (D). Considerable calcification is present (E).

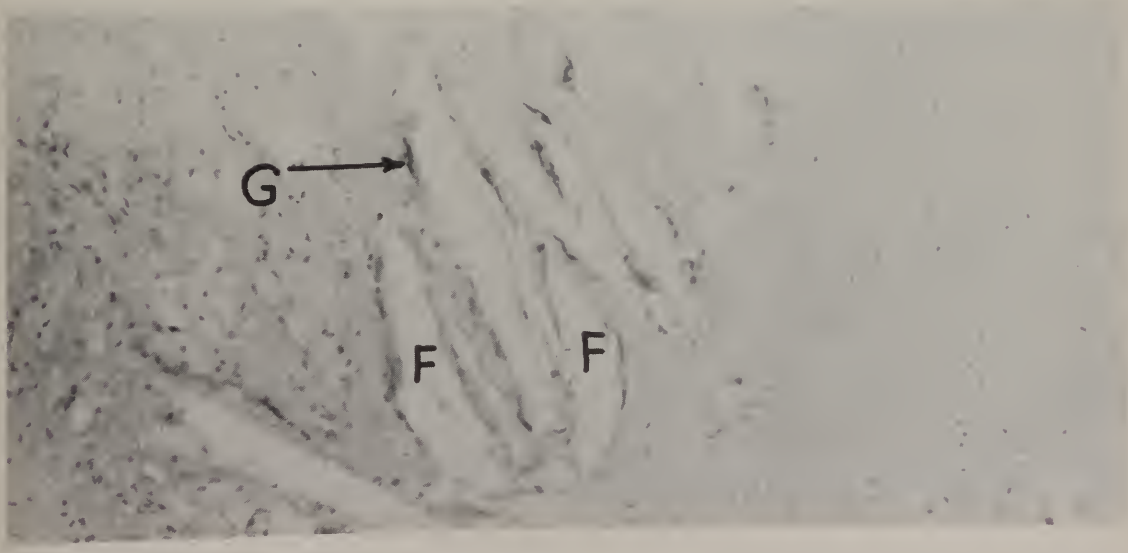
Adjacent to the tumor is dura. Its dense collagenous connective tissue is edematous. Parts of it are degenerated. Within the dura there are elongated empty crevices (F) which were previously the sites of cholesterol deposits; their immediate walls contain multinucleate giant cells (G).

This architecture of the tumor suggests adamantinoma.

Reference: Critchley, McD., and Ironsides, R. N.: The pituitary adamantinomata, Brain 49: 473, 1926.

SLIDE 69. CRANIOPHARYNGIOMA





NEG. 72838a X 75 NEG. 72838b X 810 NEG. 72996 X 200

MENINGIOMA (MENINGOTHELIAL TYPE)

Hematoxylin and Eosin Stain

CLINICAL NOTE: Not available. (MP 3071)

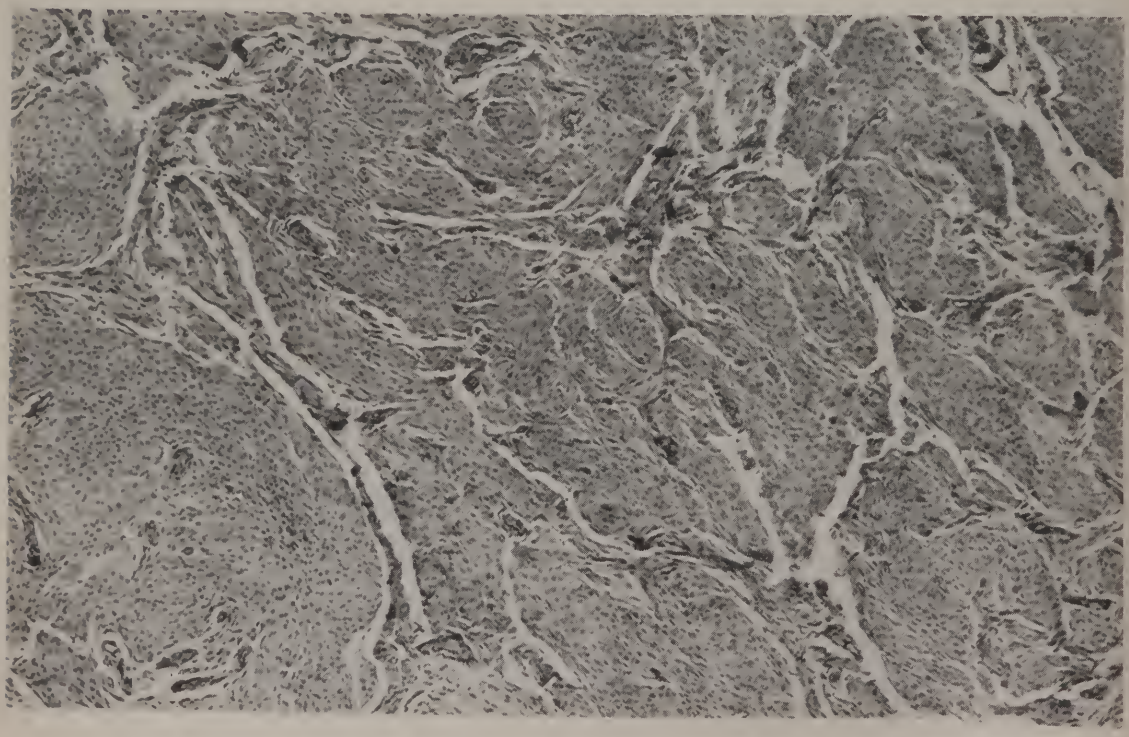
PATHOLOGY: The section shows dura mater invaded by tumor. The latter is poorly vascularized and is composed of masses of cells separated by narrow fibrous strands. The arrangement is reminiscent of alveoli.

The neoplastic cells are of the meningothelial type: they are closely packed and are fairly uniform in size and appearance, and although their shapes vary, the majority are polyhedral. The nuclei are relatively pale and vesicular. The cytoplasm is fairly abundant.

There is a tendency to form whorls which in places have concentric onion-layer-like centers. There are, however, no calcium-containing concentric structures: i.e., no psammoma body formation.

Other varieties of meningioma are shown in Slides 71, 72 and 84.

Reference: Penfield, W.: Tumors of the sheaths of the nervous system. In Cytology and cellular pathology of the nervous system, New York, Hoeber, 1932, vol. 3, pp. 959-967.



NEG. 72828 X 75

ANGIOBLASTIC MENINGIOMA

Hematoxylin and Eosin Stain

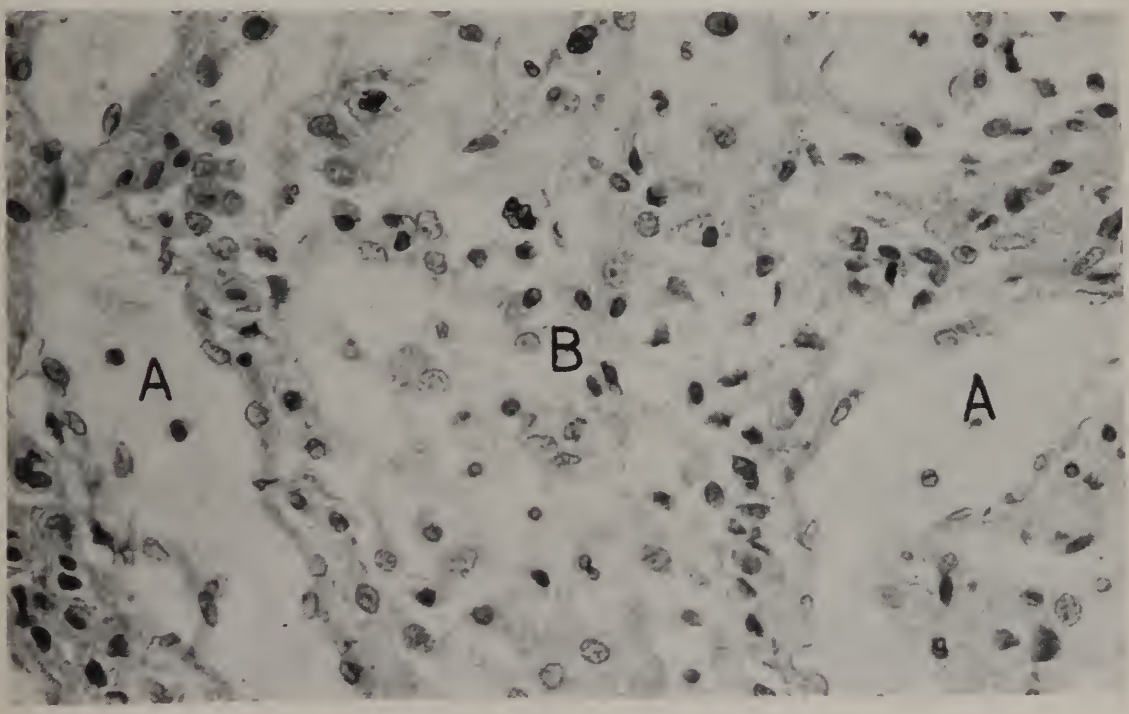
CLINICAL NOTE: A 47-year-old female who had symptoms of a rapidly growing brain tumor. The onset occurred about two or three months prior to operation. (A 3606)

PATHOLOGY: The tumor, soft and hemorrhagic, was parasagittal in location and compressed the left parietal lobe.

The section shows an exceedingly vascular tumor, the vessel walls being disarranged and chaotic (A). The adventitia of many of the vessels is considerably thickened and here and there somewhat homogeneous. The stroma of the tumor is loculated and contains many cell shapes, of which the spindle and polyhedral forms predominate (B)

In short, the tumor consists of both angiomatous and meningothelial elements. (Slide 72 is from the same tumor.)

Reference: Bailey, P., Cushing, H., and Eisenhardt, L.: Angioblastic meningiomas, Arch. Path. 6: 953, 1928.



NEG. 72831 X 500

Slide 71

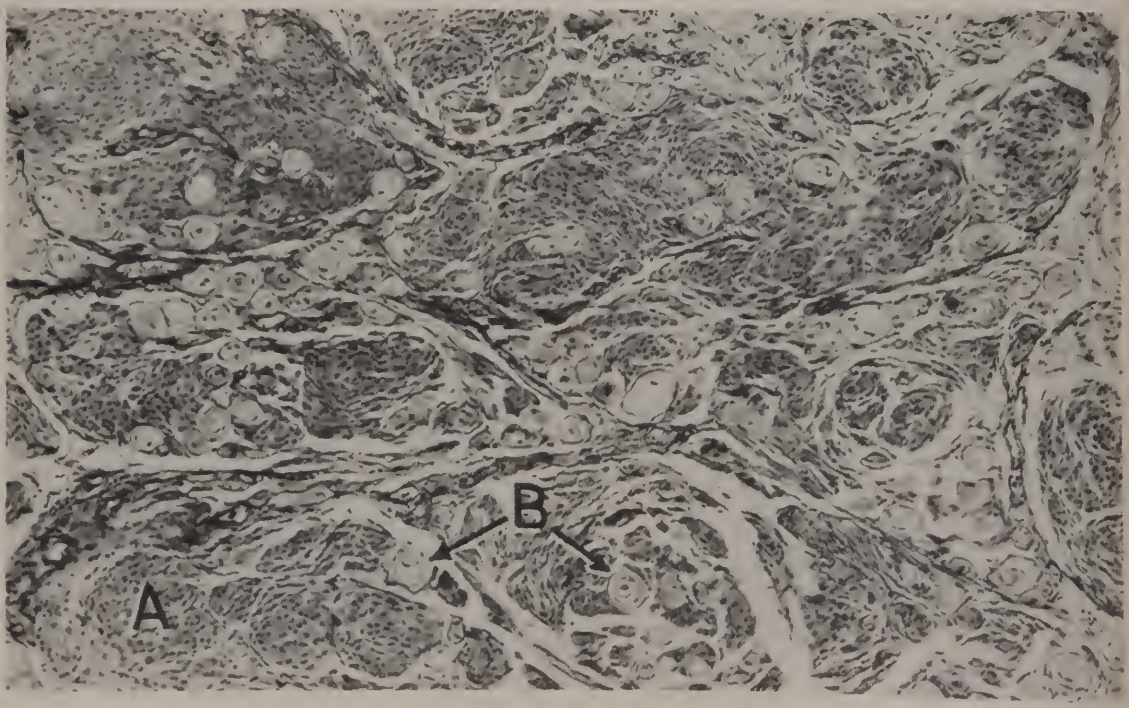
MENINGIOMA WITH WHORL FORMATION

Hematoxylin and Eosin Stain

CLINICAL NOTE: Same case as Slide 71. (A 3606)

PATHOLOGY: This portion of the tumor had invaded the skull. The section shows interlacing compact bundles of spindle-shaped or polyhedral cells with rather vesicular nuclei and abundant cytoplasm. Throughout the section the cells have taken on the pattern of whorls (A). Many of the whorls are arranged in concentric lamellae (B). The central portions of some of the lamellae contain small deposits of calcium. Clear-cut psammoma, or arenaceous, bodies are not present.

Reference: Alpers, B. J., and Grant, F. C.: Primary fibroblastoma of the brain, Arch. Neurol. & Psychiat. 27: 270, 1932.



NEG. 72837 X 75

Slide 72

SUBACUTE COMBINED DEGENERATION OF THE SPINAL CORD

Loyez Myelin Sheath Stain

CLINICAL NOTE: Not available.

PATHOLOGY: The section is from the thoracic part of the cord, which is the portion most commonly affected in subacute combined degeneration. The most conspicious change is in the lateral and dorsal columns, where "status spongiosis" exists.

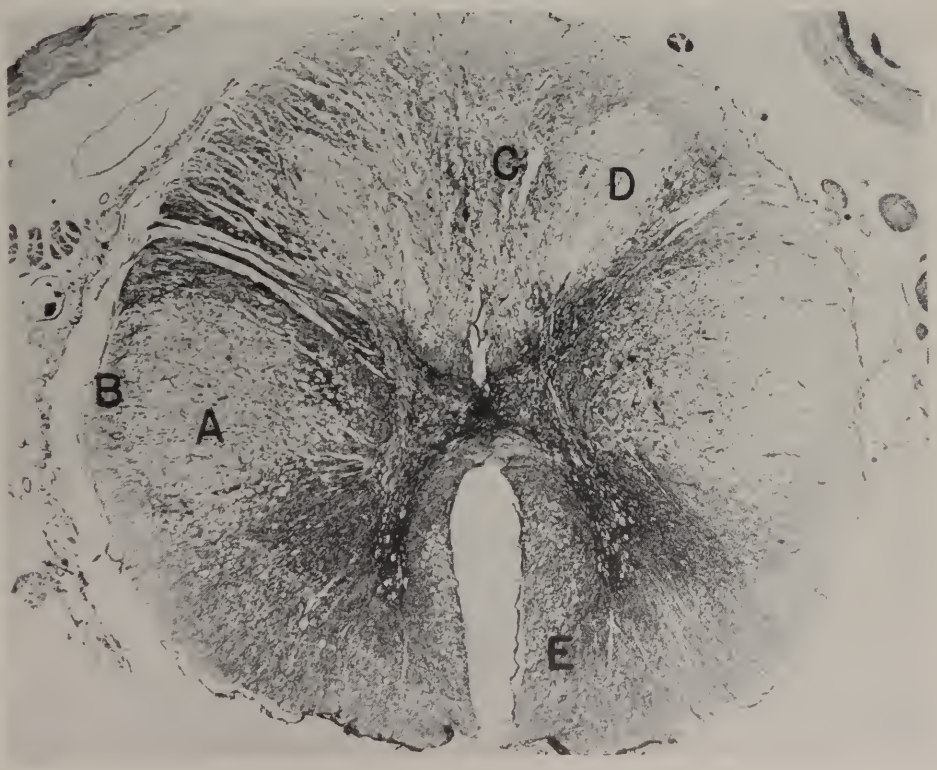
In the lateral column the pyramidal tract (A) is virtually devoid of myelin. The bordering dorsal spinocerebellar tract (B) is less affected.

In the dorsal column the gracile (C) and cuneate (D) tracts suffer to about the same degree, but usually the former is more subject to demyelination than the latter (see Slide 50). The portion of the cuneate tract adjacent to the dorsal horn is well preserved, a feature not encountered in tabes dorsalis (see Slide 5). The dorsal root is moderately demyelinated.

In the ventral column the anterior, or uncrossed, pyramidal tract is partly demyelinated (E).

Although the anterior horn cells cannot be seen to good advantage they are intact in this disorder.

Reference: Weil, A., and Davison, C.: Changes in the spinal cord in anemia, Arch. Neurol. & Psychiat. 22: 966, 1929.



Slide 73 NEG. 76612 X 14

HYDROCEPHALUS

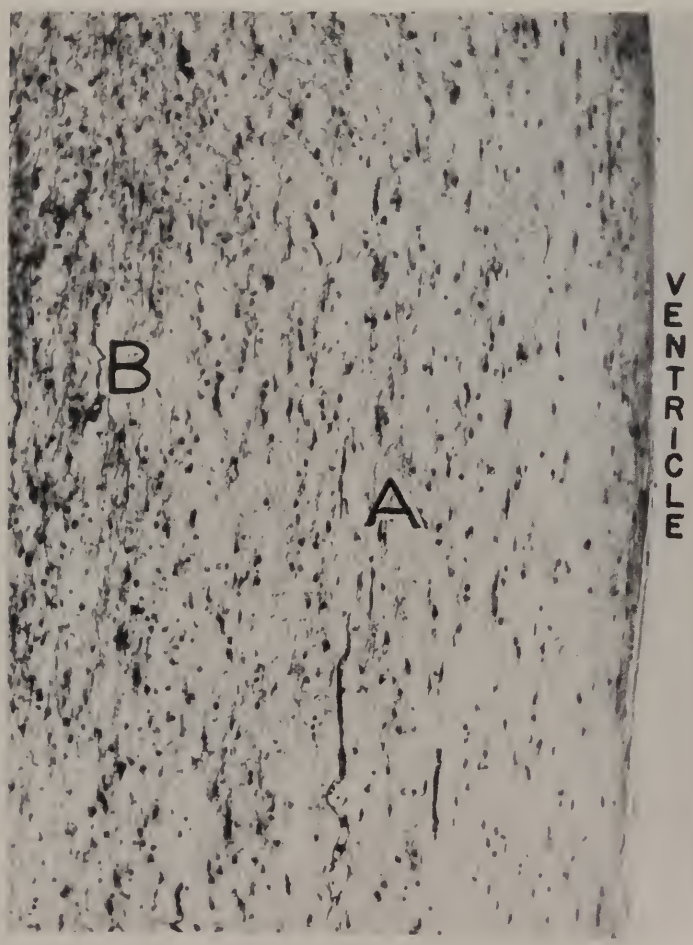
Loyez Myelin Sheath Stain

CLINICAL NOTE: Not available.

PATHOLOGY: The section is through the wall of a hydrocephalic lateral ventricle. Virtually none of the ependymal lining remains. The part of the wall bordering the ventricle is composed largely of dense interwoven wavy fibers (neuropil) running parallel to the ventricular surface.

Demyelination is visible in the more compressed region of the ventricular wall (A); in more distant parts the myelin is relatively intact (B). A few perivascular hemorrhages are noted in the vicinity of the ventricle. Virtually all the reactive cells are astrocytes.

Reference: Penfield, W., and Elvidge, A. R.: Hydrocephalus and the atrophy of cerebral compression. In Penfield, W., Cytology and cellular pathology of the nervous system, New York, Hoeber, 1932, vol. 3, pp. 1203-1217.



NEG. 72857 X 150

Slide 74

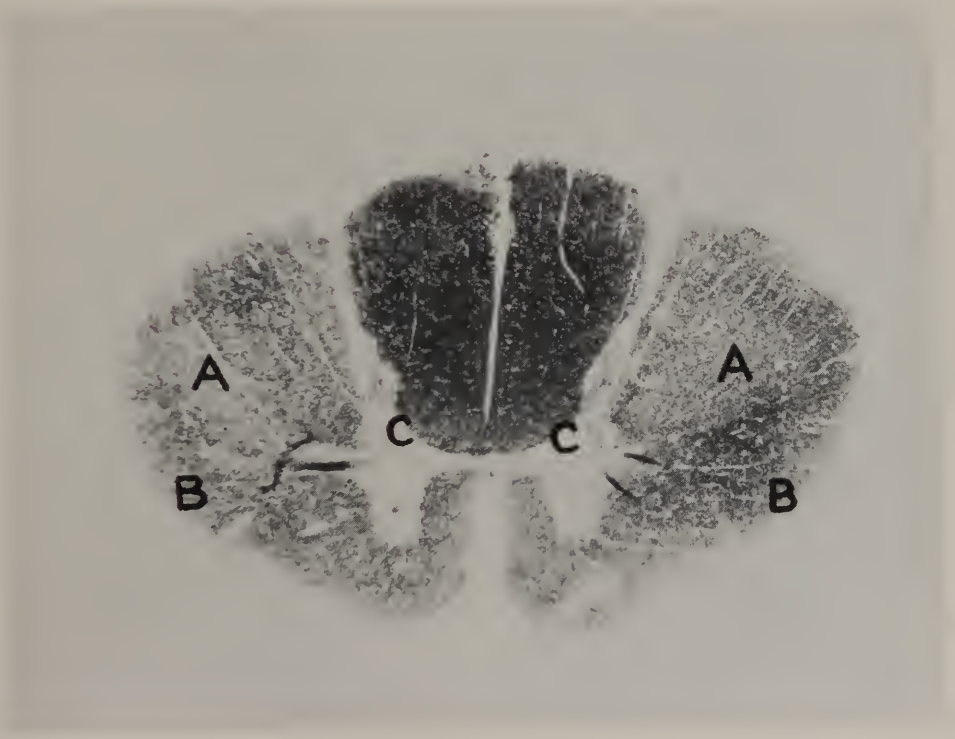
AMYOTROPHIC LATERAL SCLEROSIS

Spielmeyer Myelin Sheath Stain

CLINICAL NOTE: A female, 62 years old, with a typical clinical picture of amyotrophic lateral sclerosis. (NP 204)

PATHOLOGY: Demyelination is particularly evident in the pyramidal tracts (A). It is also present throughout the lateral and ventral columns, but to a lesser degree. The demyelination in the anterior spinocerebellar tracts (B) is consistent with atrophy of the nucleus dorsalis (C). Many of the cells of the anterior horns have disappeared. (See also Slide 87, which is stained by the Nissl Method.)

Reference: Wechsler, I. S., and Davison, C.: Amyotrophic lateral sclerosis with mental symptoms, Arch. Neurol. & Psychiat. 27: 859, 1932.



NEG. 72833 X 10

Slide 76

THE TEMPORAL CORTEX AND HIPPOCAMPAL FORMATION IN A CASE OF ACUTE YELLOW ATROPHY OF THE LIVER

Nissl Stain

CLINICAL NOTE: A male, 20 years old, who had marked jaundice of two weeks duration. Terminally there were generalized epileptiform seizures. (A 3740)

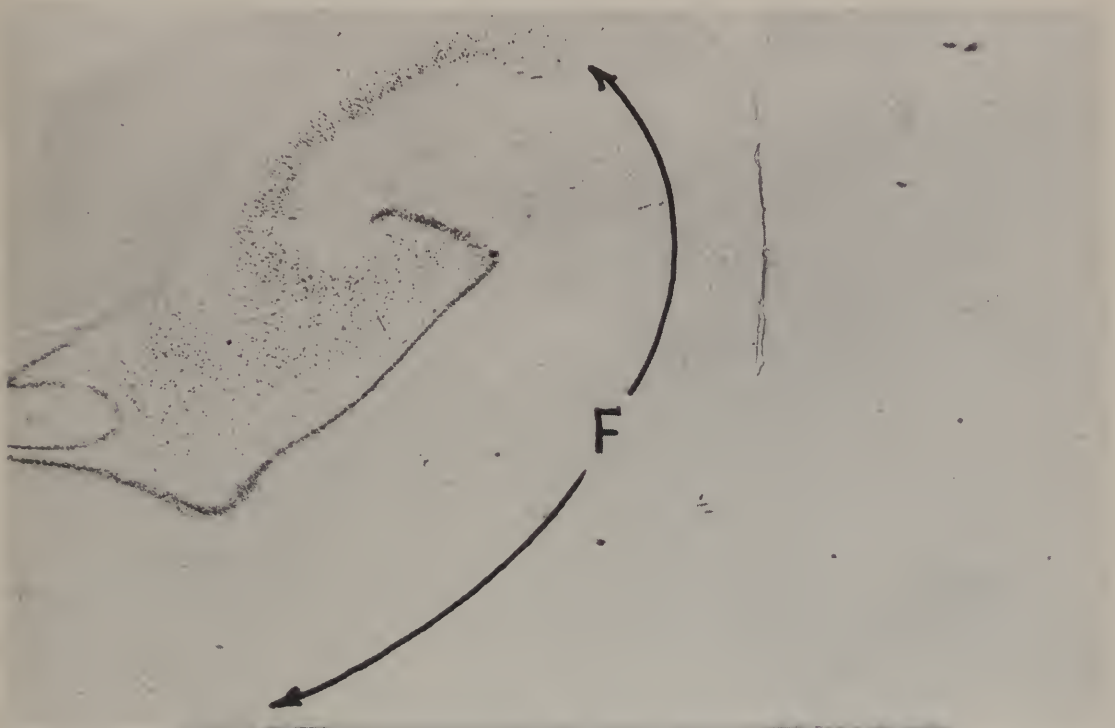
PATHOLOGY: The liver showed typical acute yellow atrophy. No gross changes in the brain were noted.

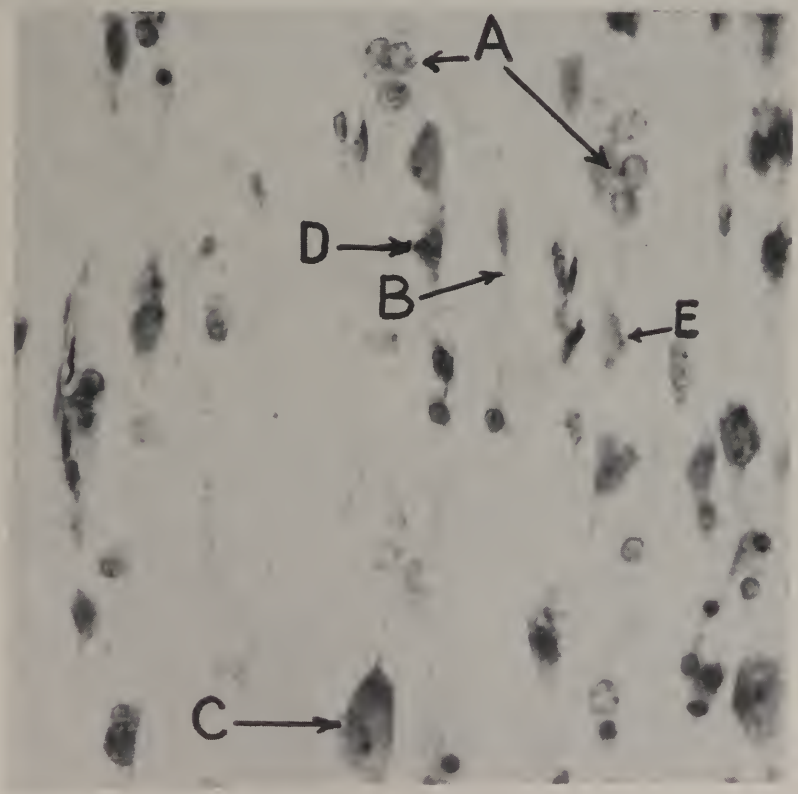
The section is interesting in two respects. Throughout the cortex there are numerous astrocytes with characteristically pale, rather large nuclei (A) as well as linear microglia resembling rod cells (B). Some of the ganglion cells are swollen and have eccentric nuclei (C); others are pyknotic (D) while still others are ghost-like (E). Such changes are said to be frequent in acute or chronic liver disease.

The other point of interest is in the hippocampus where much of the pyramidal layer is devoid of ganglion cells (F). Early necrosis is present in those cells that remain. These changes are ascribed to anoxia associated with the terminal epileptiform seizures.

Reference: Weil, A., and Crandall, L. A.: The pathology of the central nervous system in liver disease, Trans. Am. Neurol. Ass'n., 1932.

SLIDE 77. THE TEMPORAL CORTEX AND HIPPOCAMPAL FORMATION IN A CASE OF ACUTE YELLOW ATROPHY OF THE LIVER





NEG. 72883a X 15 NEG. 72883b X 650

POLIOMYELITIS

Nissl Stain

CLINICAL NOTE: Not available.

PATHOLOGY: Most of the cells of the anterior horn of the right side of the spinal cord have disappeared (A). Remaining cells show varying degrees of chromatolysis, shrinkage and other degenerative changes (B). The circle of oligodendrocytes about some of the more degenerated cells suggests that neuronophagia may still be underway. Throughout both anterior horns, particularly the one on the right there is a diffuse increase of astrocytes and microglia. The same applies to the posterior horns, but to a lesser degree. Gliosis is also visible throughout the white matter.

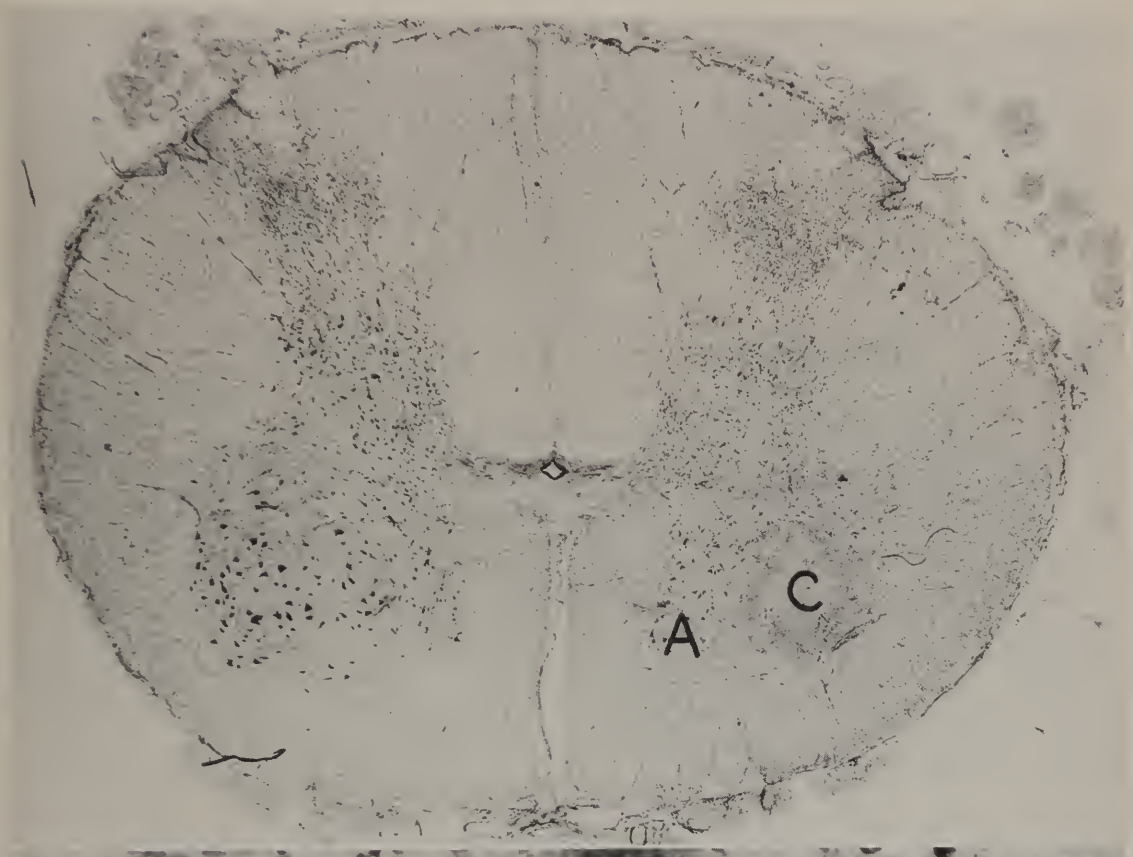
An old focus in the right anterior horn has been transformed into a scar (C) consisting mostly of astrocytes. The leptomeninges are somewhat thickened and congested and contain a few inflammatory cells.

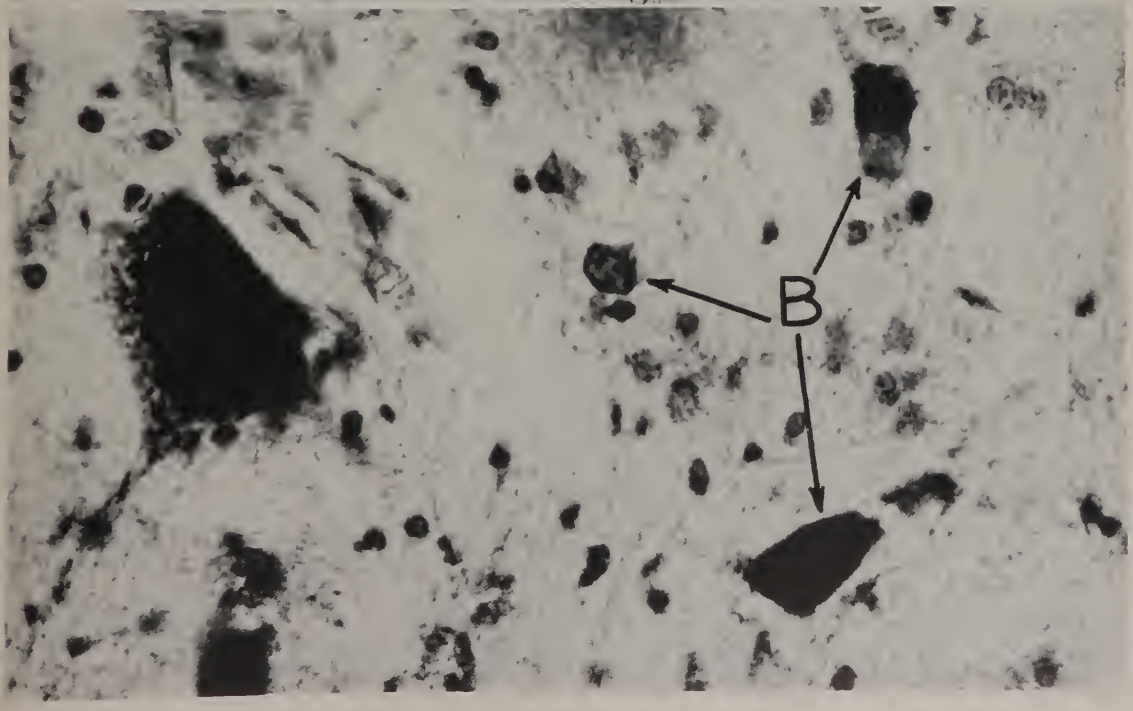
The acute phase of the disease is past and reparative processes are in full swing.

References: Hurst, E. W.: The histology of experimental poliomyelitis, J. Path. & Bact. 32: 457, 1929.

Smith, N. R.: Acute anterior poliomyelitis, Brit. Med. J. 2: 476, 1929.

SLIDE 78. POLIOMYELITIS





NEG. 72905 X 15 NEG. 73836 X 705

RECURRENT GLIOMA

Nissl Stain

CLINICAL NOTE: A 42-year-old female had a tumor removed from the right parietal lobe. Diagnosis at that time was astrocytoma. For a while she was relatively symptom-free but six months after operation there was a recurrence of symptoms; three months later she died. (MP 3799)

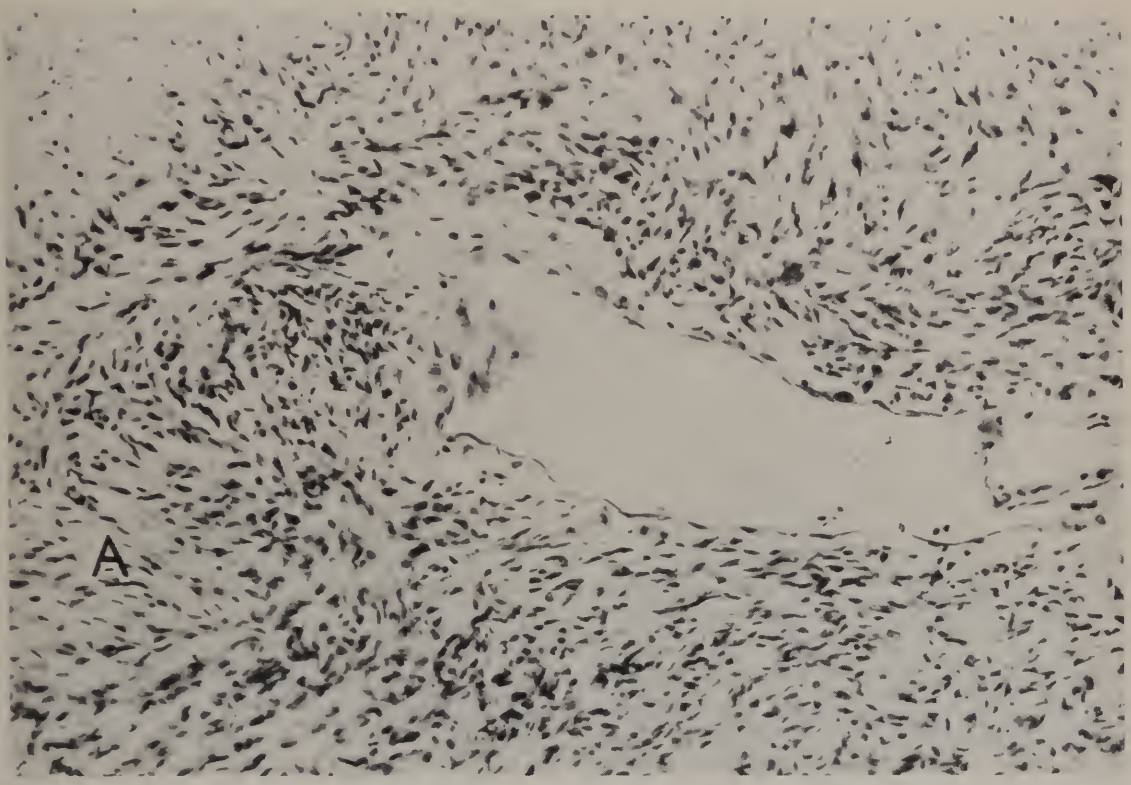
PATHOLOGY: At the site of the old operation there was a cystic tumor about the size of an orange.

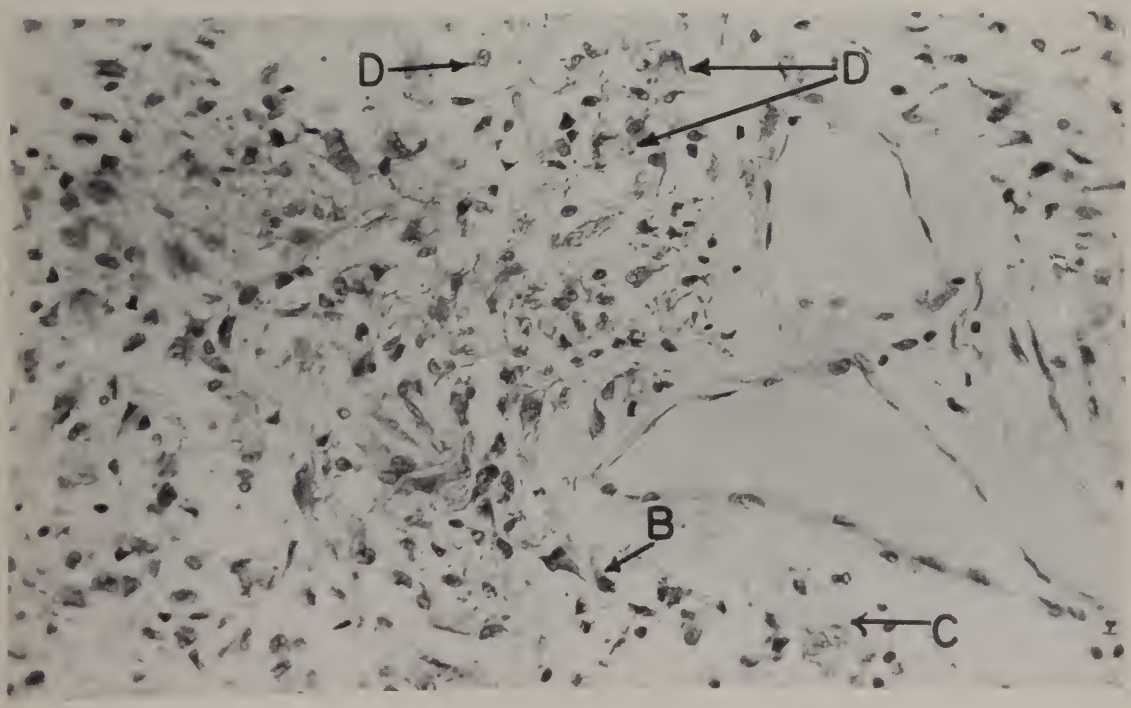
A portion of the tumor consists of cells with elongated nuclei and tail-like processes which are grouped together in criss-crossing fascicles (A). The architecture of the major part of the section is lacking in uniformity, there being many cells with thick polar processes (B), a few cells with star-shaped cytoplasm (C), and a moderate number of cell forms with bizarre misshapen nuclei (D).

The tumor is moderately vascular and contains a number of large cysts. Necrotic areas are common: some of them contain numerous leukocytes.

Owing to the pleomorphism, this tumor may at first glance be regarded as a glioblastoma multiforme but since the elongated astrocyte is the chief cell form the tumor is regarded as a pilocytic astrocytoma. Not inconsistent with this diagnosis are the pleomorphism and the cyst formation.

SLIDE 79. RECURRENT GLIOMA





NEG. 72898 X 200 NEG. 73890 X 350

OLIGODENDROGLIOMA

Hematoxylin and Eosin Stain

CLINICAL NOTE: A 43-year-old woman who for 15 months had had frequent epileptic seizures. A tumor of the right fronto-parietal region was partially removed. Two days later death occurred. (A 3427)

PATHOLOGY: The tumor is composed of a variety of cell forms and has architectural features that vary somewhat from field to field. The dominant cell form is small and has a spherical nucleus which is either vesicular (A) or dense (B). Clear spaces, or halos, surround the nuclei; in such spaces one notes a sparse finely granular cytoplasm. Such cells, particularly those with dense nuclei, are regarded as neoplastic oligodendrocytes. Some of the vessels contain calcium deposits (C), which are also a feature of oligodendroglioma.

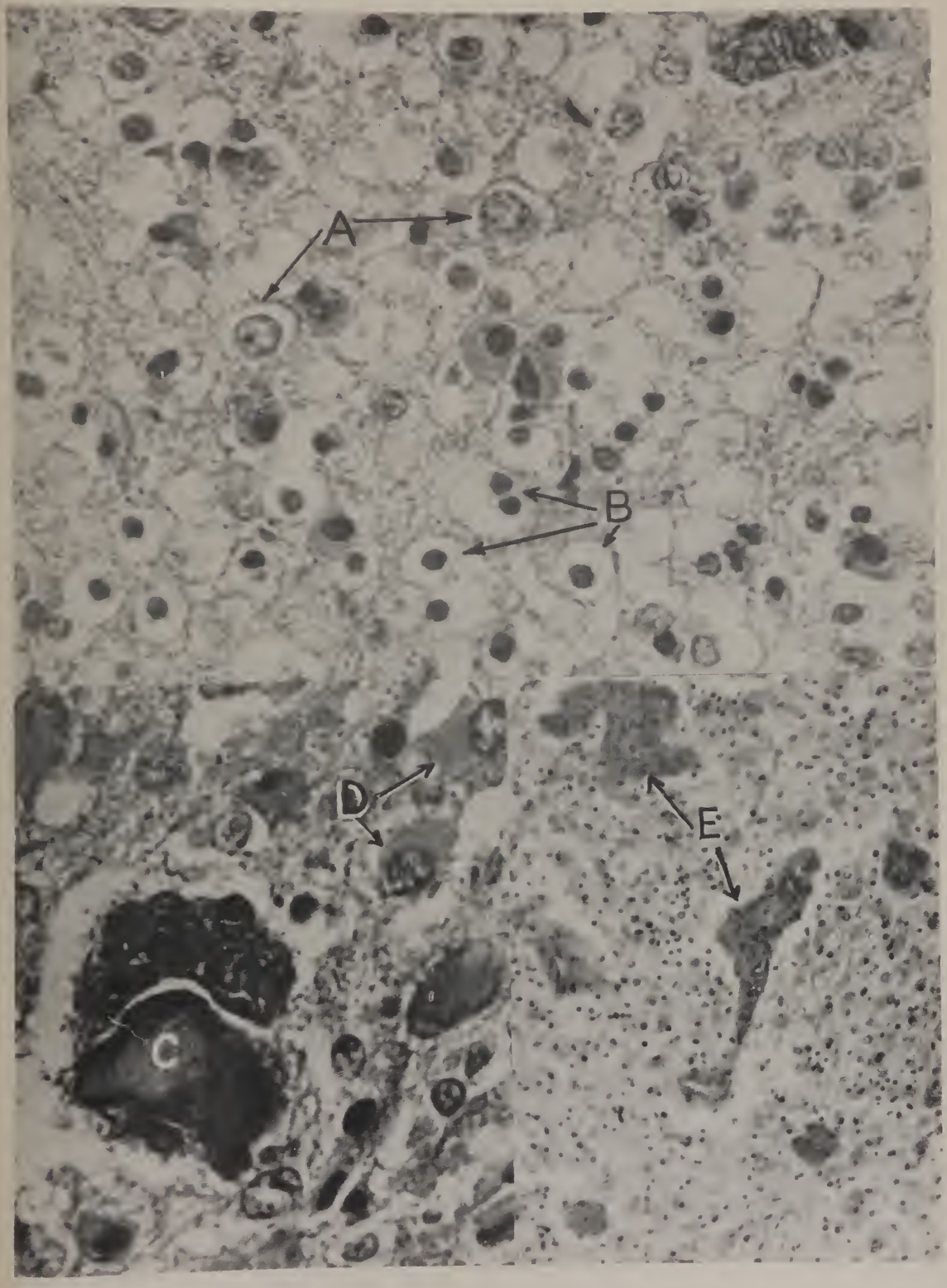
There are also numerous cells with abundant homogeneous cytoplasm and vesicular nuclei (D). Such cells are regarded as gemistocytic astrocytes.

Another feature is the presence of neoplastic (or hyperplastic) blood vessels (E). These are most commonly encountered in glioblastoma multiforme.

Taken as a whole the section is regarded as an oligodendroglioma with a trend toward astrocytoma. From the assortment of cell forms and the abnormal vessel formations in this section it would not be surprising if other sections showed a more frank indication of glioblastoma multiforme.

Reference: Elvidge, A., Penfield, W., and Cone, W.: The gliomas of the central nervous system, A. Research Nerv. & Ment. Dis., Proc. 16: 107, 1935.

SLIDE 80. OLIGODENDROGLIOMA



NEG. 72899 X 810 NEG. 74046 X 1000 NEG. 74042 X 175

ASTROCYTOMA DIFFUSUM

Nissl Stain

CLINICAL NOTE: A 38-year-old female who for several years had shown symptoms of a slowly growing tumor of the right frontal lobe. (A 3422)

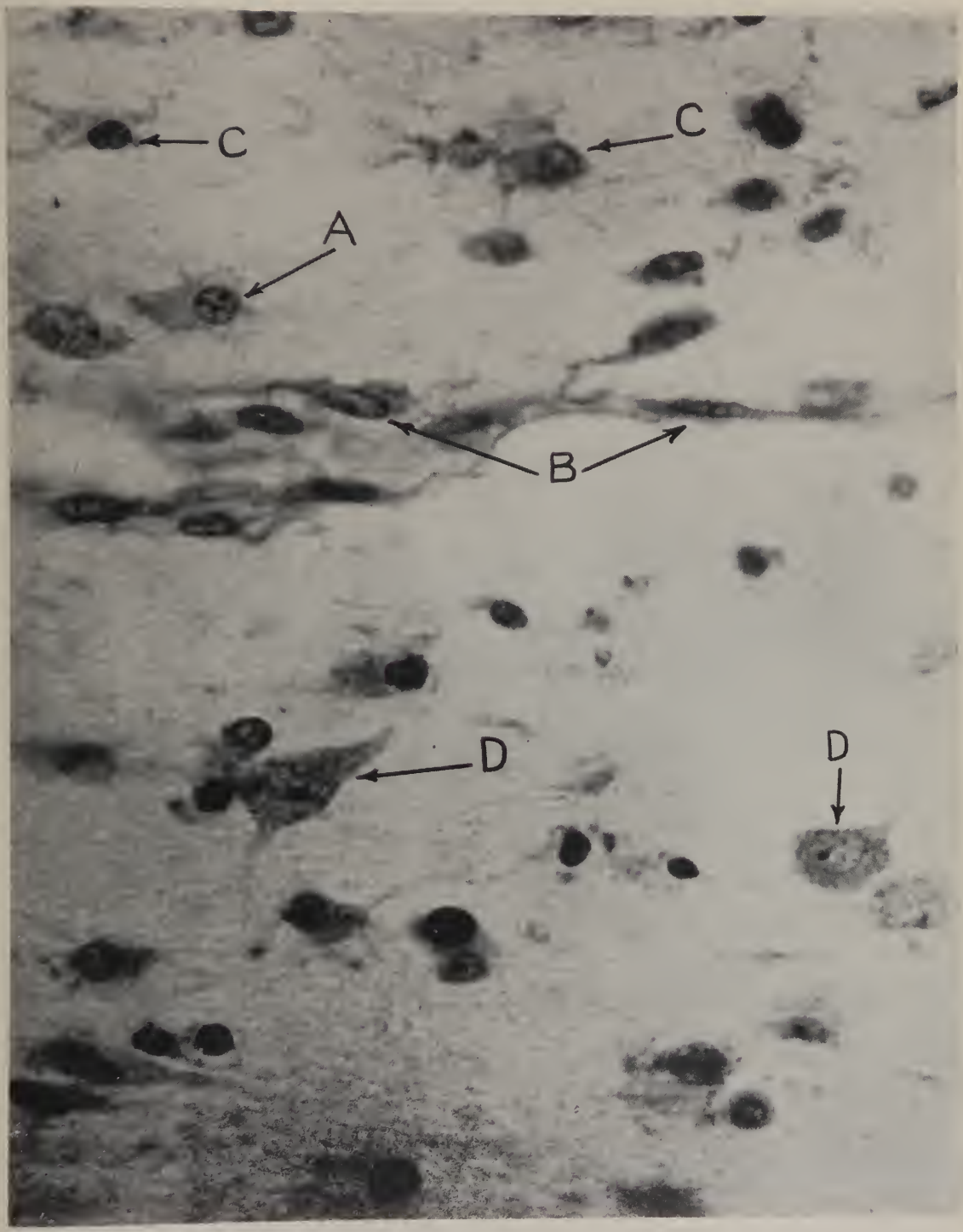
PATHOLOGY: The tumor was grossly similar to that of the previous case (Slide 81) but less gelatinous and of a firmer consistency.

Individual tumor cells invade diffusely the cerebral cortex. In lower cortical layers one notes the perineuronal satellitosis characteristic of astrocyma diffusum. On the whole the cells are poorly demarcated and sparse. A variety of astrocytic forms is noted: scattered plump cells with moderate sized vesicular nuclei, cells having a somewhat triangular shape (A), cells with long trailing cytoplasmic processes such as one finds in the pilocytic type of astrocytoma (B), and relatively mature cells having the appearance of protoplasmic astrocytes (C). The nerve cells (D) are probably inclusions. Vascularization is sparse.

The poor demarcation of the cells, the diffuse invasion and the perineuronal satellitosis mark this as an astrocytoma diffusum.

Reference: Greenfield, J. G.: The pathological examination of forty intracranial neoplasms, Brain 42: 29, 1919.

SLIDE 82. ASTROCYTOMA DIFFUSUM



NEG. 74047 X 1000

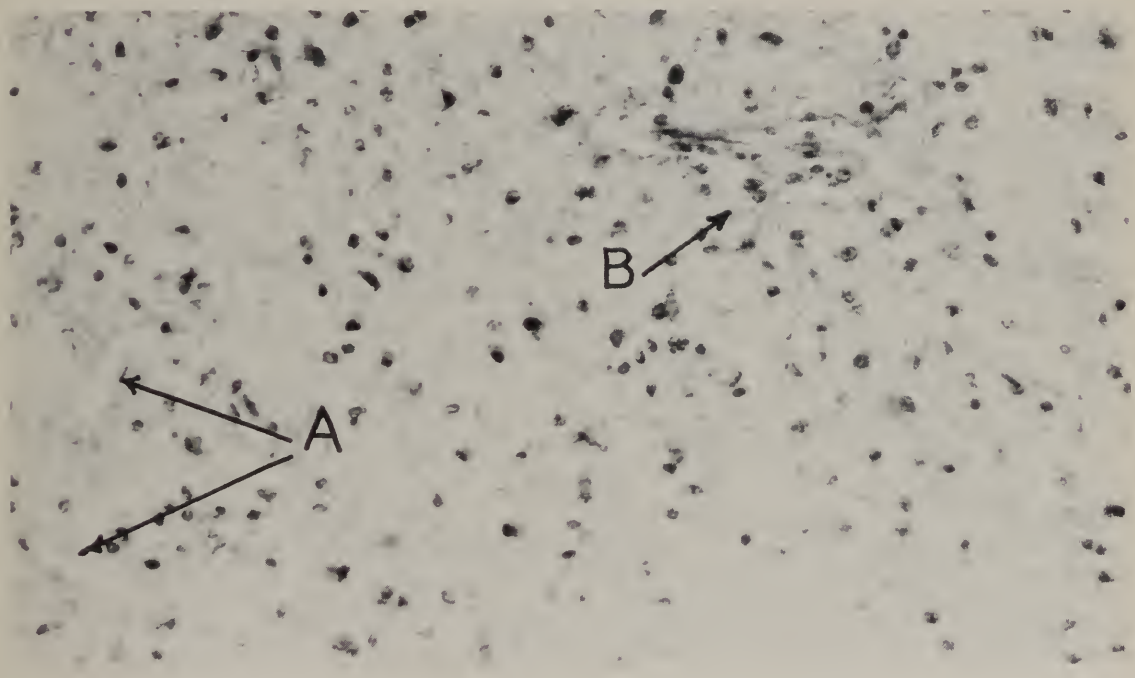
ASTROCYTOMA, PILOCYTIC TYPE

Phosphotungstic Acid and Hematoxylin Stain

CLINICAL NOTE: A child with symptoms of a cerebellar tumor.

PATHOLOGY: Adjacent to the tumor one finds the remains of cerebellar folia. The tumor itself contains numerous cyst-like spaces which vary considerably in size, some of the smallest being illustrated in the photograph (A). Vascularity is moderate (B). A dense feltwork of glial fibers is a conspicuous feature of the tumor. So marked is the proliferation of glial processes that it is impossible to determine the cells to which many of the individual fibers belong. From the poles of some of the cells there extend rather broad single processes, to which the cell type owes its name (pilus, hair; cyte, cell). The nuclei of the astrocytic forms are of moderate size and vesicular.

Reference: Elvidge, A., Penfield, W., and Cone, W.: The gliomas of the central nervous system, A. Research Nerv. & Ment. Dis., Proc. 16: 107, 1937.



NEG. 72902 X 300

Slide 83

GLIOMA, PROBABLE EPENDYMOMA

Hematoxylin and Eosin Stain

CLINICAL NOTE: A 20-month-old girl who became progressively weak and soon was unable to walk. Tremor was a predominant symptom. Operation disclosed a tumor surrounding the medulla oblongata and filling the cisterna magna. It is not clear from the description that the tumor involved the medulla oblongata. (SP 19371)

PATHOLOGY: Microscopically the tumor is uniform throughout. It contains large numbers of blood vessels (A), each surrounded by a relatively wide zone of fibrillar tissue (B). Most of the fibrils are so fine that they are individually indiscernible but here and there one sees broad fibers extending inward to become anchored in the vessel wall. The cells from which the fibers originate are piled up at the periphery of the fibrillar zone.

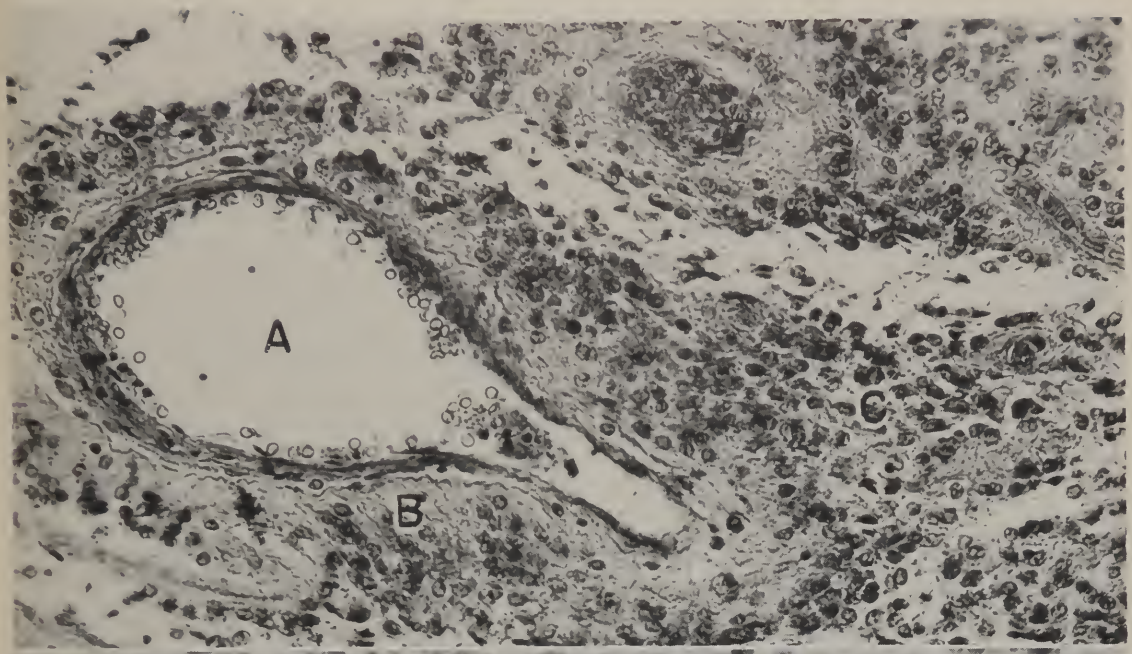
The intervascular tissue (C) is fairly cellular. The nuclei are spherical and vesicular and the cytoplasm is fairly abundant. Here and there one notes minute acellular fibrillar areas bordered by cell bodies, a layer or more in thickness (pseudo-rosettes).

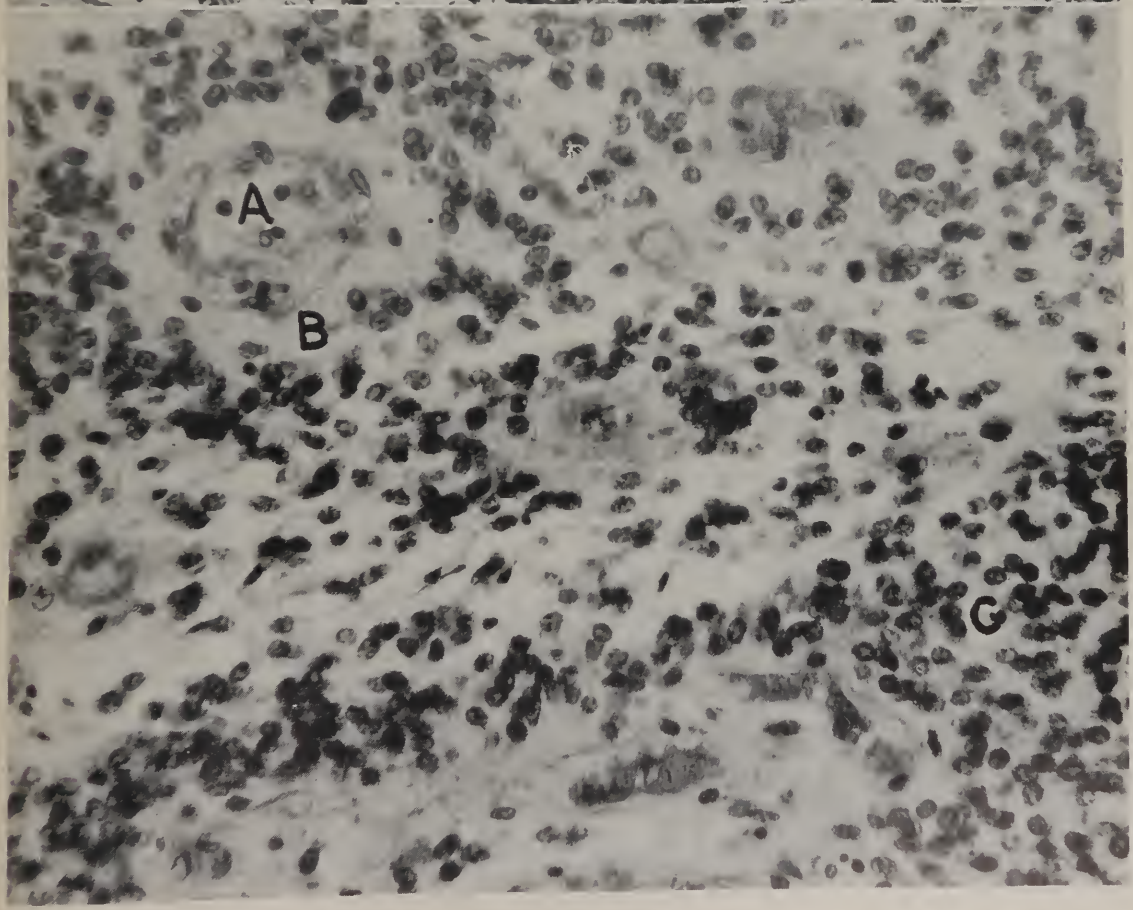
This slide has been passed to various workers in the tumor field for an opinion as to diagnosis. The radiation of processes in the form of "feet" and the vesicular nature of the nuclei have been taken by three of the consultants as evidence for astroblastoma but it seems to the writers that the processes are much too fine and interwoven to be those characteristic of astroblastoma. The fact that the tumor was mainly in the meninges suggested to another consultant that this is a tumor of the perithelial tissue of meningeal blood vessels, hence a meningioma, but although this argument has its points, the vessels of angioblastic meningiomas are rarely if ever surrounded by a feltwork of fibers. From the pseudo-rosettes and the nature of the cells as well as the general architecture, three other persons concluded that this is an ependymoma. The balance of evidence seems to favor the latter diagnosis even though there are more blood vessels than are usually encountered in ependymoma and even though the characteristic colloid-like areas are absent.

References: Hashimoto, S.: Zur Kenntnis der Zylindrome und Peritheliome des Gehirns, Arb. a.d. neurol. Inst. a.d. Wien. Univ. 29: 357, 1927.

Kernohan, J. W., and Fletcher-Kernohan, E. M.: Ependymomas, A. Research Nerv. & Ment. Dis., Proc. 16: 182, 1935.

SLIDE 84. GLIOMA, PROBABLE EPENDYMOMA





NEG. 73892 X 355 NEG. 76619 X 460

PINEALOMA

Hematoxylin and Eosin Stain

CLINICAL NOTE: A 19-year-old male who had symptoms of a rapidly growing brain tumor. Internal hydrocephalus was advanced. (A 3299)

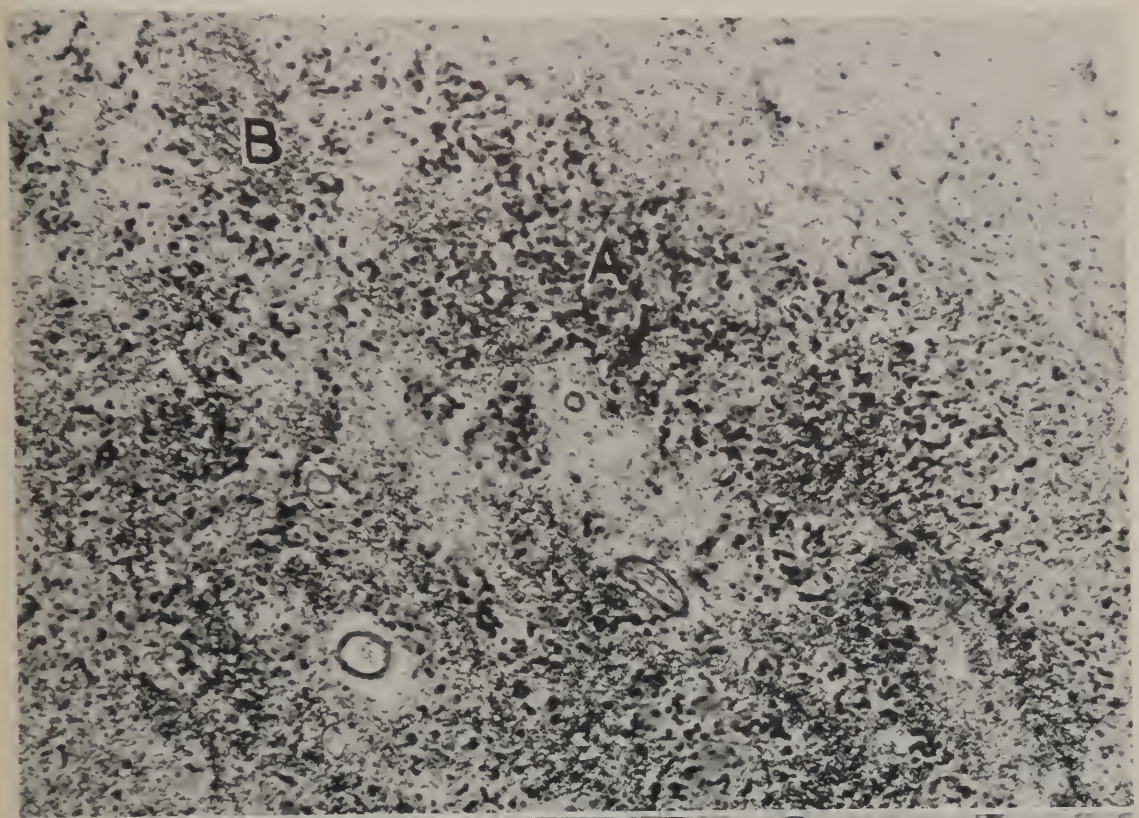
PATHOLOGY: Autopsy disclosed a small tumor in the roof of the IIId ventricle which had occluded the aqueduct.

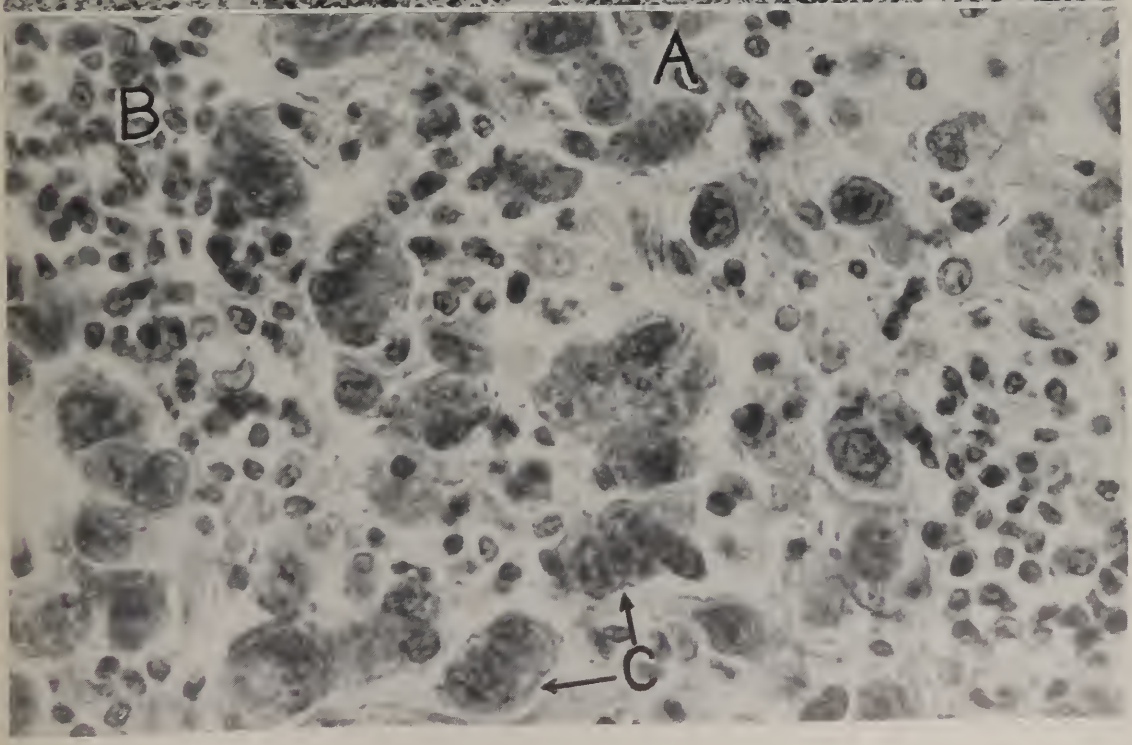
Histologically the tumor is moderately vascularized and consists of groups of large cells (A) scattered indiscriminantly in a stroma of lymphoid tissue (B). Here and there multinucleate cells (C) are noted. Cells undergoing mitosis are also present. The nuclei of the tumor cells are large, variously shaped, vesicular, and contain one or two distinct nucleoli.

Connective tissue septa replete with the lymphoid cells, ordinarily a feature of this tumor, are not well seen in this instance.

Reference: Horrax, G., and Bailey, P.: Tumors of the pineal body, Arch. Neurol. & Psychiat. 13: 423, 1925.

SLIDE 85. PINEALOMA





NEG. 72889a X 75 NEG. 72889b X 705

HEMANGIOBLASTOMA OF THE CEREBELLUM

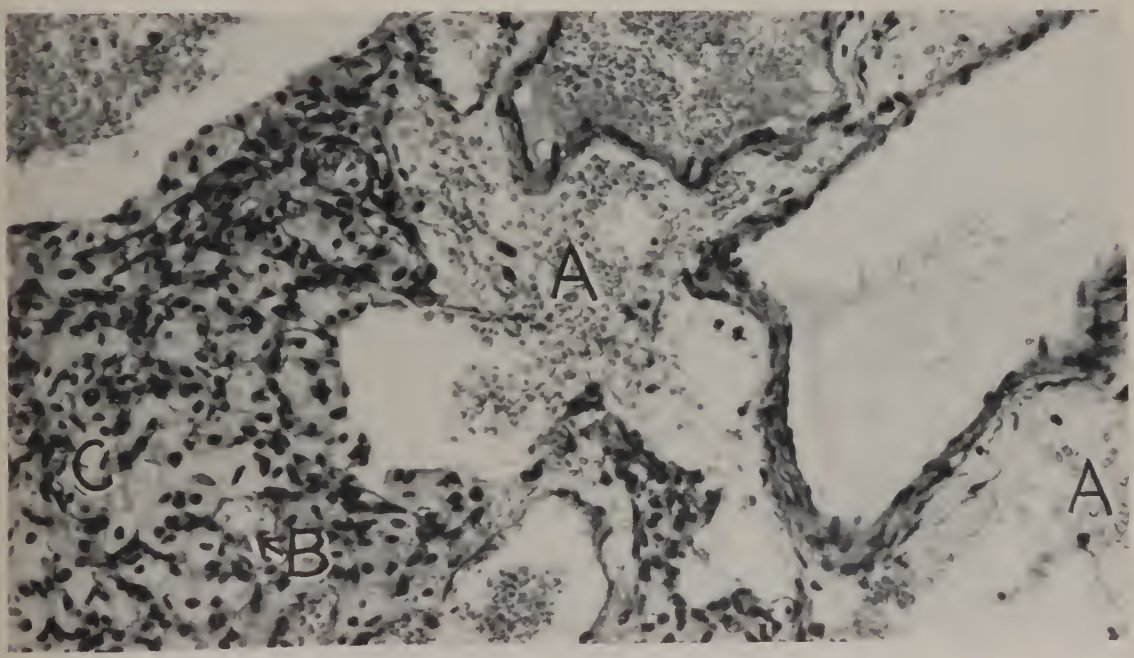
Hematoxylin and Eosin Stain

CLINICAL NOTE: A 47-year-old male who had unmistakable signs of a cerebellar tumor. At operation the cerebellar tumor was completely excised. Several years previously a "hemangioma" had been removed from the retina. (NP 112)

PATHOLOGY: A part of the section contains cerebellar folia which are heavily strewn with red blood cells. The tumor is composed of wide cavernous spaces filled with blood (A), and of myriads of anastomosing capillaries (B). Lying between the blood spaces are cells of diverse size and shape with relatively abundant homogeneous or finely vacuolated cytoplasm (C). Pigment-laden macrophages are present at the edge of the section. Some parts of the tumor have undergone necrosis.

This is a mixed cavernous and capillary hemangioblastoma characteristic of von Hippel-Lindau's disease. Usually this tumor appears as a small nodule on the wall of a large cyst.

Reference: Cushing, H., and Bailey, P.: Blood vessel tumors of the brain, Springfield, Ill., C. C. Thomas, 1928, pp. 108-186.



NEG. 73837 X 375

Slide 86

AMYOTROPHIC LATERAL SCLEROSIS

Nissl Stain

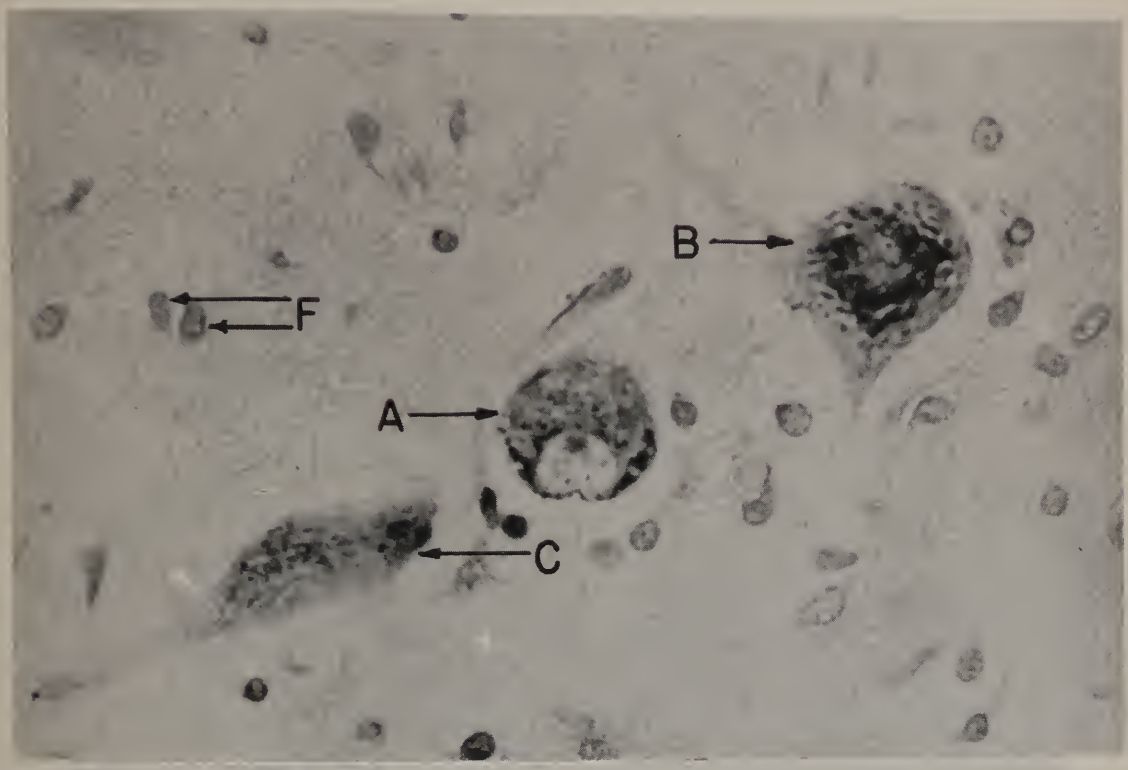
CLINICAL NOTE: A 23-year-old woman who over a period of eighteen months had developed spasticity of the lower extremities and atrophy in the small muscles of the hands. Signs of bulbar involvement ensued and respiration became embarrassed. For several months she was kept in a respirator. The immediate cause of death was bronchopneumonia. (A 3600)

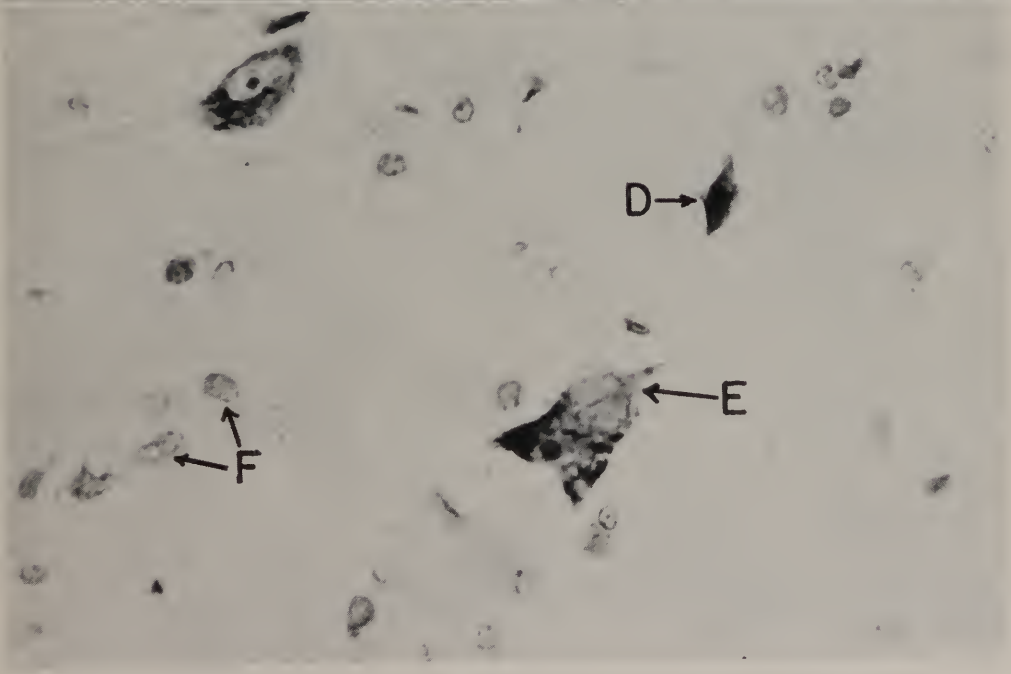
PATHOLOGY: The anterior horns of the spinal cord are markedly deficient in nerve cells. Some of those remaining have undergone considerable degeneration: chromatolysis (A), swelling (B), eccentric displacement of nucleus and nucleolus (C), shrinkage (D), local ballooning of cytoplasm (E). The small cells scattered throughout the anterior horn are mostly astrocytes (F).

Owing to the stain employed, the degeneration of the pyramidal tracts is not visible. Such changes are shown in Slide 76.

Reference: Weil, A.: A text-book of neuropathology, Philadelphia, Lea & Febiger, 1933, p. 228.

SLIDE 87. AMYOTROPHIC LATERAL SCLEROSIS





NEG. 72891 X 810 NEG. 73976 X 600

THE BRAIN IN A CASE OF ECLAMPSIA OF PREGNANCY

Nissl Stain

CLINICAL NOTE: A 29-year-old woman had had an uneventful pregnancy until convulsive seizures set in. Following the eighth convulsion she died. (A 3886)

PATHOLOGY: At autopsy, normally developed full-term twins were found. The kidneys were the seat of nephrosis and the liver showed periportal necrosis. The brain was grossly normal.

The section is through the temporal cortex and adjoining hippocampal formation. The lamination of the temporal cortex appears somewhat disorganized owing to the distortion of individual nerve cells and the presence of proliferated microglia.

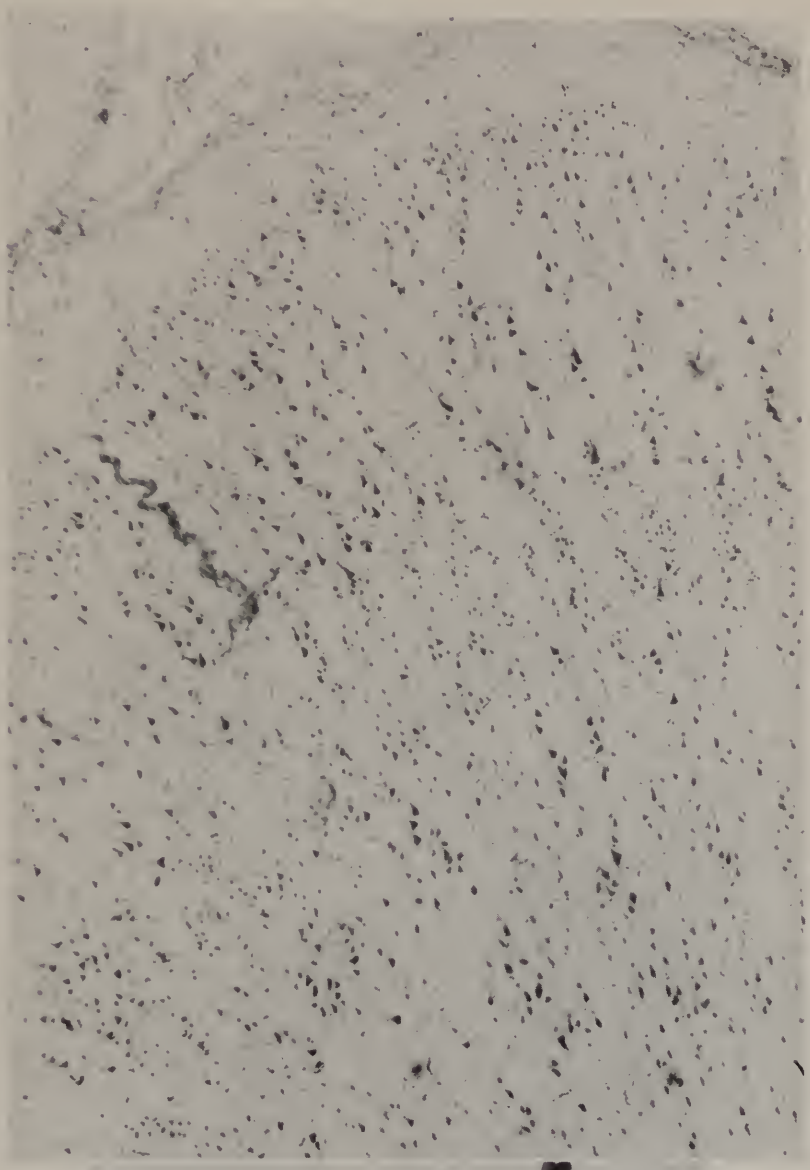
The ganglion cells show various degrees of degeneration. A striking feature is the tortuosity of apical dendrites. The cytoplasm of some of the ganglion cells is spongy and pale; the nuclei are mostly eccentric and pyknotic. In the lower layers of the cortex there are satellitosis and neuronophagia (A). (Similar changes are present in the pyramidal layer of the hippocampus).

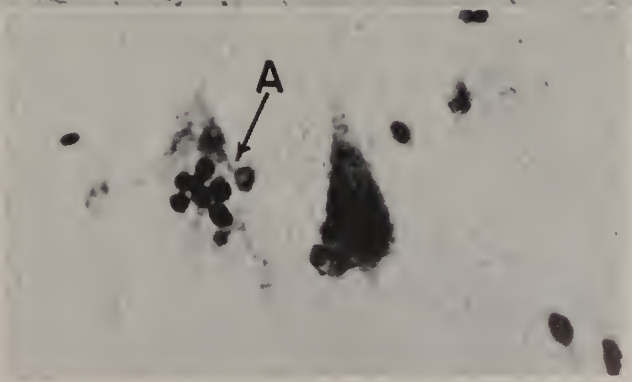
As to the proliferated microglia permeating the temporal cortex, they are characterized by a small, flattened, almost linear cell body, with thin processes radiating from either end. They were doubtless present before the occurrence of the convulsive seizures.

Note: Some of the sections labeled Slide 88 are from another part of the cerebral cortex. Here there is increased vascularity and a marked replacement of ganglion cells by astrocytes and microglia. The white matter also has undergone gliosis.

Reference: Hassin, G. B.: Histopathology of the peripheral and central nervous systems, ed. 2, New York, Hoeber, pp. 252-254.

SLIDE 88. THE BRAIN IN A CASE OF ECLAMPSIA OF PREGNANCY





NEG. 73027 X 80 NEG. 73977 X 680

MEDULLOBLASTOMA

Hematoxylin and Eosin Stain

CLINICAL NOTE: A boy, 4 years old, had had headache and vomiting for four months. Neurological examination disclosed evidence of tumor of the left cerebellar hemisphere. The optic discs were considerably choked. An Attempt at removal of the tumor was unsuccessful, death occurring post-operatively. (NP 102)

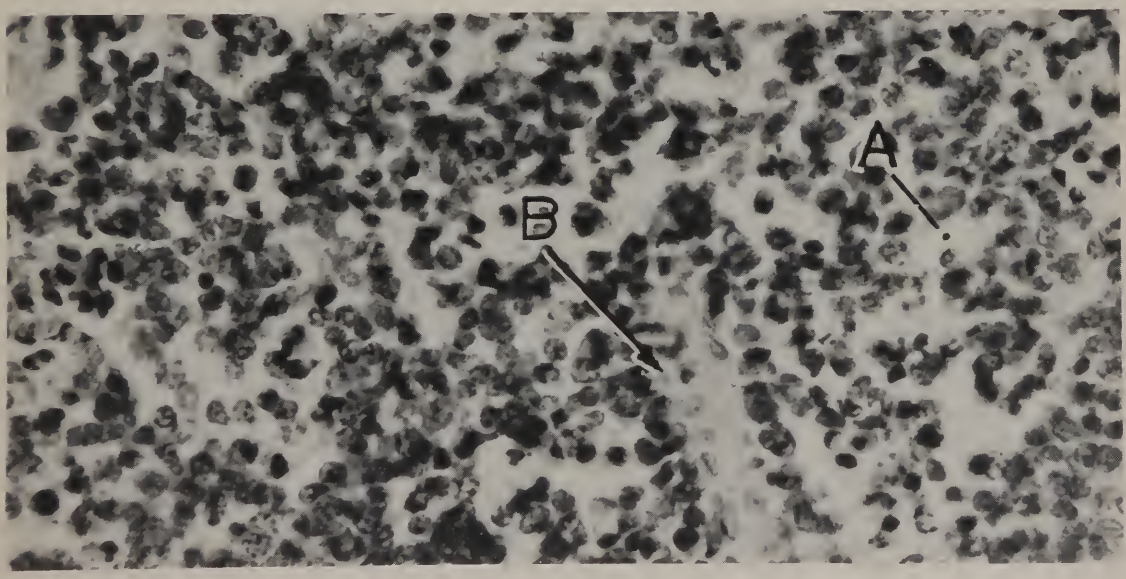
PATHOLOGY: Autopsy disclosed a large tumor of the cerebellum involving the corpora quadrigemina, and obliterating the aqueduct. The pineal region and the thalami were also encroached on.

The tumor is composed of masses and cords of densely packed small cells with scanty cytoplasm and dark-staining spherical or ovoid nuclei. An occasional pseudo-rosette is seen: This consists of a ring of cells enclosing a structureless core (A). (In ependymoma, by way of contrast, the core consists of fine processes radiating inward).

Outstanding in this section is the clustering of tumor cells about blood vessels (B). (In ependymoma the cells are separated from the blood vessels by a broad seam of neuropil whereas in this tumor the cells cells hug the vessels.)

References: Kershman, J.: The medulloblasts and the medulloblastoma, Arch. Neurol. & Psychiat. 40: 936, 1938.

Elvidge, A., Penfield, W., and Cone, W.: The gliomas of the central nervous system, A. Research Nerv. & Ment. Dis., Proc. 16: 107, 1937.



NEG. 74559 X 550

Slide 89

CORTICAL NECROSIS IN ARTERIOSCLEROSIS

Nissl Stain

CLINICAL NOTE: A 67-year-old male who had had long-standing hypertension with evidence of cerebral involvement. (NP 127)

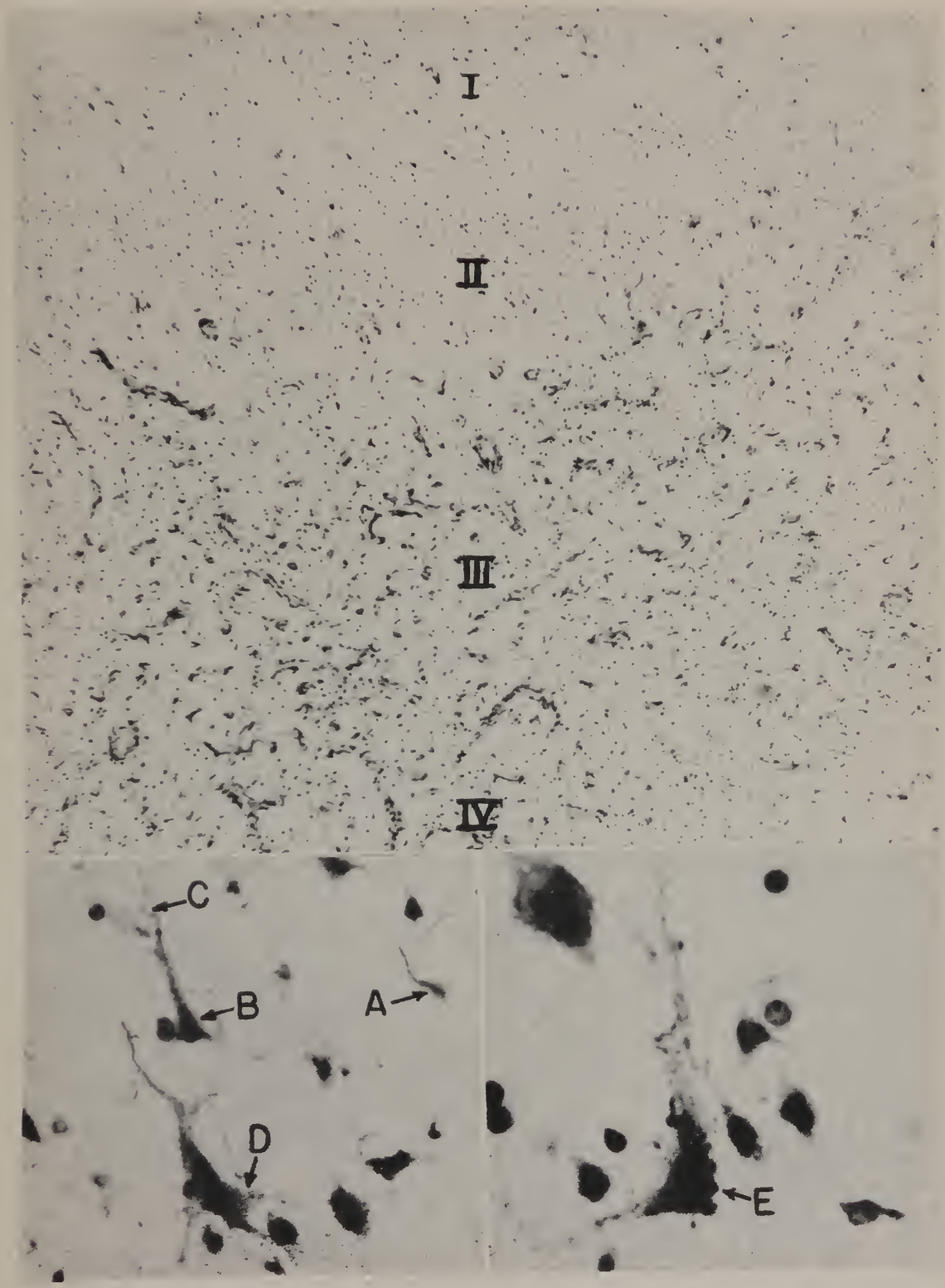
PATHOLOGY: Autopsy revealed severe sclerosis of the cerebral arteries. The brain was the seat of numerous red and white infarctions.

The photograph shows approximately four layers of the cortex, the lower two of which are virtually replaced by proliferated blood vessels and gitter and other glial cells. In layer II there is glial replacement and but little vessel hyperplasia. Layer I shows astrocytosis. Also in some of the cortical areas devoid of granulation tissue the ganglion cells have largely disappeared. Many of the ganglion cells that remain exhibit ischemic changes: shrinkage (A), pyknosis of nuclei (B), tortuosity of apical dendrites (C), distortion and thickening of processes (D) and pericellular incrustation (E). In places the lesion extends down into the white matter, as evidenced by proliferation of blood vessels, neuronophagia and perivascular accumulations of round cells.

The meshes of the pia-arachnoid are somewhat distended and contain focal collections of small round cells.

Reference: Oppenheimer, B. S., and Fishberg, A. M.: Hypertensive encephalopathy, Arch. Int. Med. 41: 264, 1928.

SLIDE 90. CORTICAL NECROSIS IN ARTERIOSCLEROSIS



NEG. 76620 X 130 NEG. 72886a X 705 NEG. 72886b X 1000

GLIOBLASTOMA MULTIFORME

Hematoxylin and Eosin Stain

CLINICAL NOTE: Not available. (NP 186)

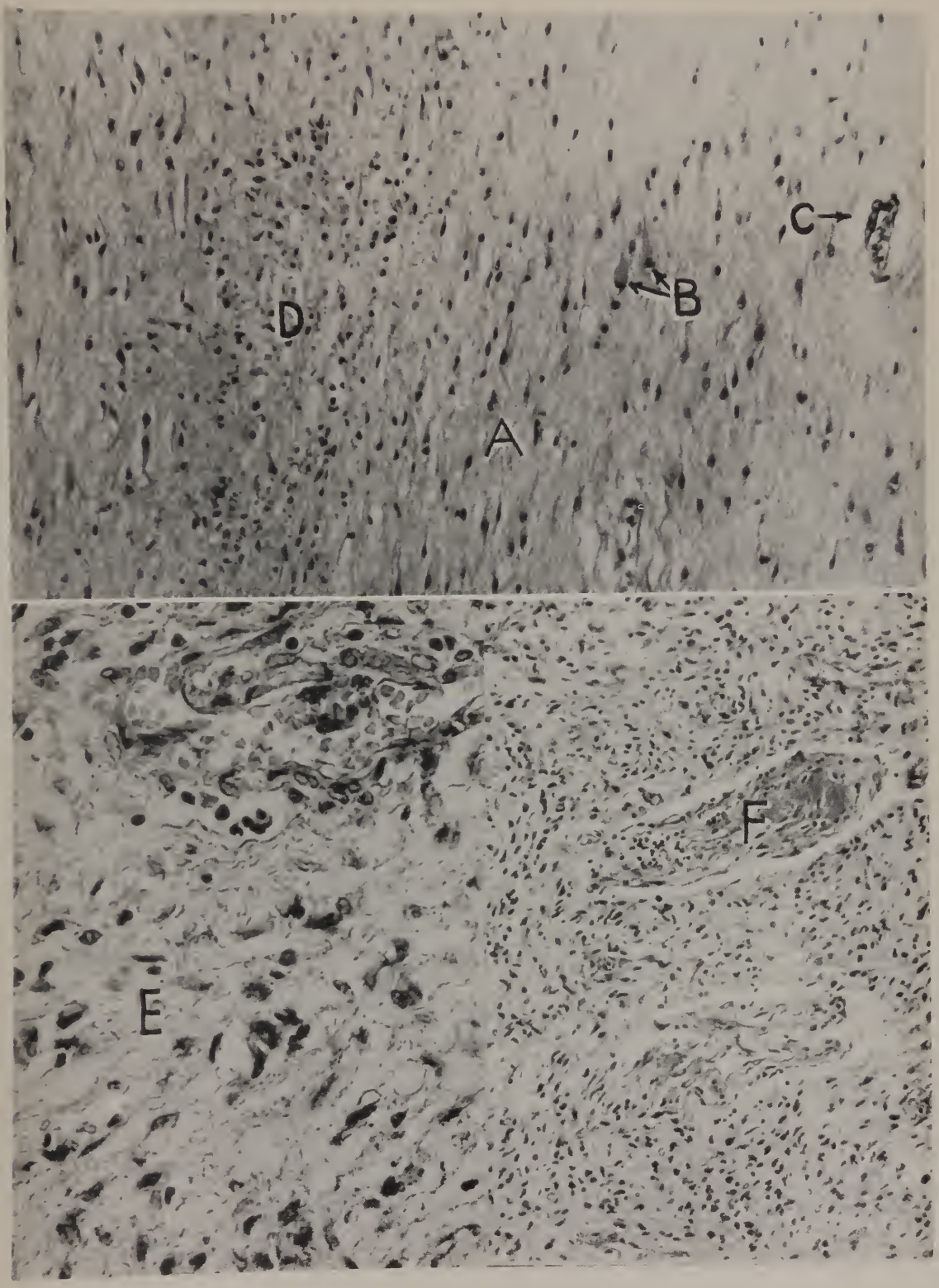
PATHOLOGY: The appearances of the section vary greatly from field to field. On one side of the section the tissue is composed of elongated astrocytes which possess somewhat wavy processes (A). Scattered among these cells are many plump astrocytes with homogeneous cytoplasm (B). The vessels (C) are few and relatively normal. Here and there one notes areas of early focal necrosis (D). Thus far the tumor conforms to astrocytoma.

The other part of the tumor is different. Here there is considerable necrosis. Such necrotic areas are bordered by rather dense groups of cells (pseudo-palisades). The viable cells vary greatly in size and appearance and have a chaotic arrangement; some of the cells are multinucleated, but the majority have the appearance of primitive spongioblasts and astrocytes (E). The walls of the blood vessels are hyperplastic and in some instances the vessels have a glomerulus-like appearance (F).

Even though there are features in some parts of the section of protoplasmic astrocytoma, taking into consideration the pleomorphism, the degeneration and the vascular changes, this tumor may be regarded as a glioblastoma multiforme.

Reference: Bailey, P.: Histologic diagnosis of tumors of the brain, Arch. Neurol. & Psychiat. 27: 1290, 1932.

SLIDE 91. GLIOBLASTOMA MULTIFORME



NEG. 72900a X 235 NEG. 72900b X 500 NEG. 74043 X 165

THE CEREBRAL CORTEX IN A CASE OF LIGHTNING STROKE

Nissl Stain

CLINICAL NOTE: An 18-year-old male who was struck by lightning and who died ten minutes thereafter. (A 3717)

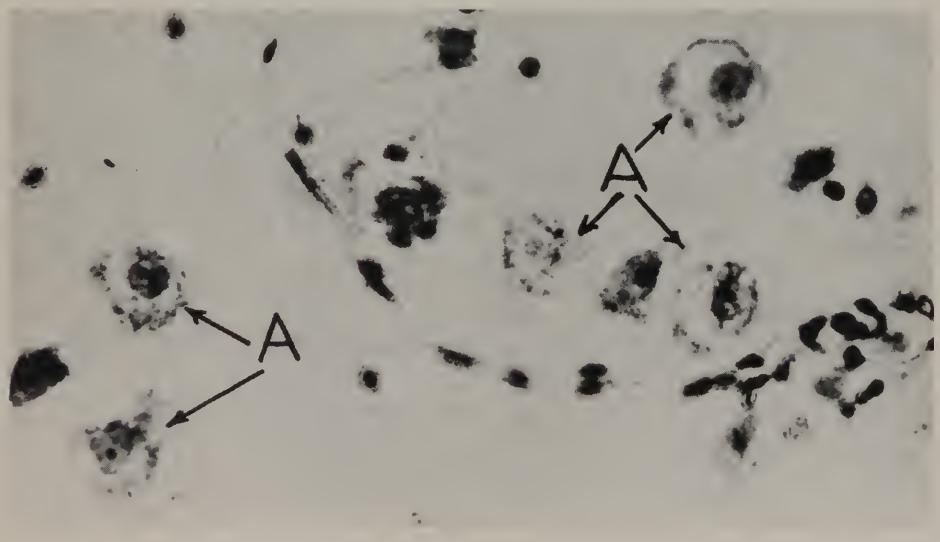
PATHOLOGY: Autopsy was performed soon after death, and the brain was fixed in 95 per cent alcohol; hence there is little possibility that the changes are artefacts. Grossly the brain looked normal.

The section is from the hippocampus and adjoining temporal cortex. Microscopically the changes are widespread. The ganglion cells show degenerative changes of varying degree. In the part of the temporal cortex photographed, many of the cells have an exploded appearance: i.e., they are ballooned and have a disrupted cytoplasm (A). In such cells the nucleus and nucleolus appear normal. In the lower layers of the cortex some of the cells are surrounded by an excess of satellite cells, which were probably present before the lightning stroke. The cells of the pyramidal layer of the hippocampus show ischemic changes.

In places the meninges are somewhat thickened and contain lymphocytes and histiocytes.

The characteristic rupture of blood vessels in lightning stroke was not seen in this section.

Reference: Hassin, G. B.: Changes in the brain in accidental electrocution, J. Nerv. & Ment. Dis. 86: 668, 1937.



NEG. 72894 X 705

Slide 92

"ETAT VERMOULU" IN CEREBRAL ARTERIOSCLEROSIS

Hematoxylin and Eosin Stain

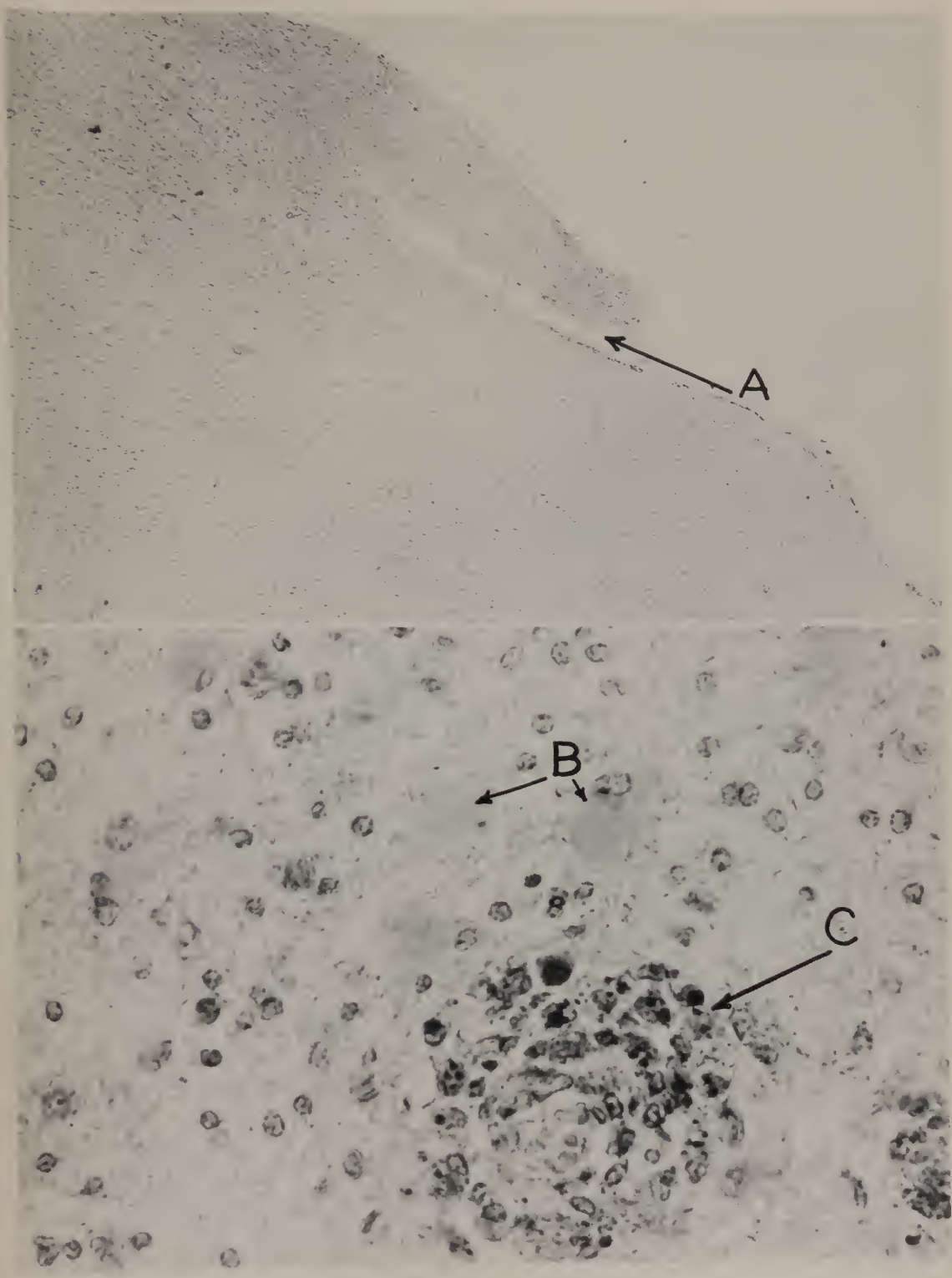
CLINICAL NOTE: A 70-year-old male who died of an otogenic meningitis. A year previously he had incurred an apparently slight cranial trauma. (NP 48)

PATHOLOGY: Grossly the left supraorbital region of the cerebral cortex showed a number of shallow irregular excavations, a sort of worm-eaten appearance (etat vermoulu).

Microscopically one of the areas in question is devoid of cortex. Adjoining cortex is undermined (A). The tissue in the sunken region is composed of swollen astrocytes (B) and other glia, and continues without boundary into relatively normal white matter. The perivascular spaces are crowded with pigment-laden gitter cells (A). The dilatations in the white matter are probably postmortem artefacts.

Reference: Hassin, G. B.: Histopathology of the peripheral and central nervous systems, ed. 2, New York, Hoeber, 1940, p. 339.

SLIDE 93. "ETAT VERMOULU" IN CEREBRAL ARTERIOSCLEROSIS



NEG. 73972 X 40 NEG. 72881 X 185

FIBRINO-PURULENT SPINAL MENINGITIS

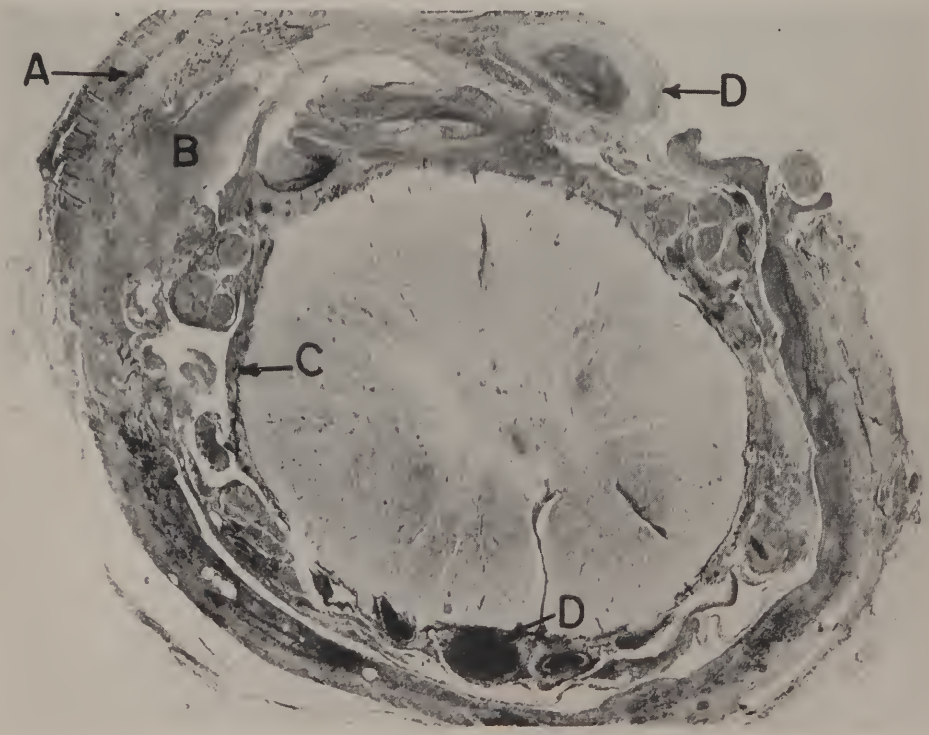
Hematoxylin and Eosin Stain

CLINICAL NOTE: Not available.

PATHOLOGY: The dura contains a few collections of gitter and other inflammatory cells (A). Contiguous with the dura there is a matrix of granulation tissue containing vast numbers of polymorphonuclear leukocytes (B). This condition is called subdural abscess or subdural empyema. The leptomeninges (C) are so greatly distended with fibrino-purulent exudate that the arachnoid trabeculae and other delicate tissues have been obliterated. Some of the vessels in the pia-arachnoid are thrombosed (D) and their walls neavily invaded with polymorphonuclear leukocytes.

In the spinal cord itself the vessels are distended. Their walls have undergone hyaline degeneration, and are infiltrated with inflammatory cells. The parenchyma in the vicinity of some of these vessels also contains inflammatory cells. Most of the anterior horn cells exhibit the acute swelling generally associated with ischemia. The wide spaces around cells represent edema. Some of the vacuoles in the white matter appear to be artifacts.

Reference: Hassin, G. B.: Histopathology of the central and peripheral nervous systems, ed. 2, New York, Hoeber, 1940, pp. 422-425.



NEG. 72896 X 6

Slide 95

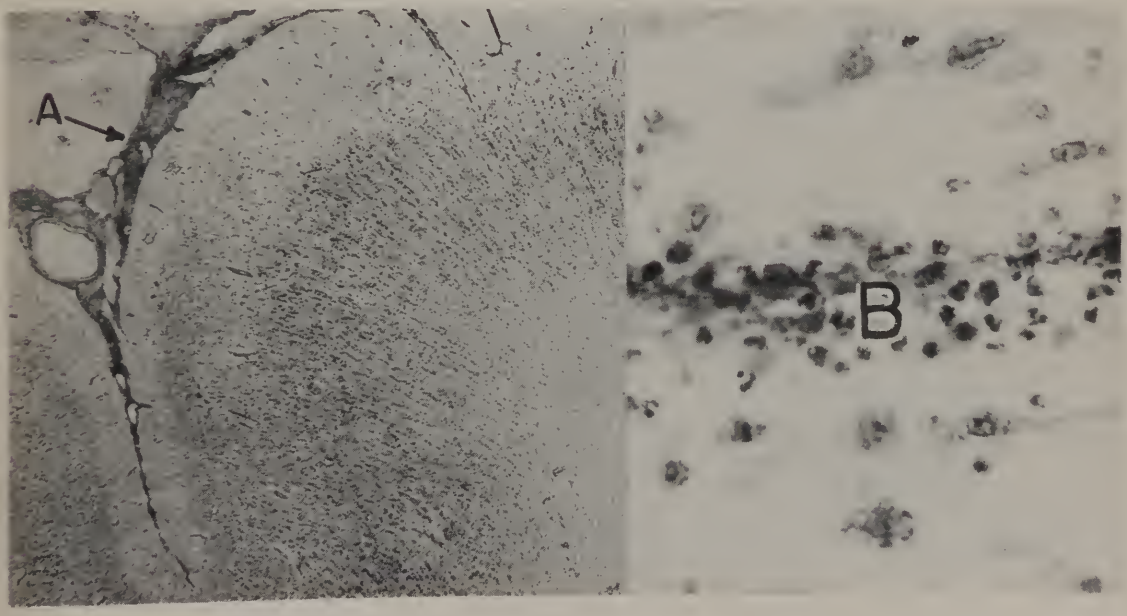
ACUTE MENINGO-ENCEPHALITIS

Nissl Stain

CLINICAL NOTE: A 14-year-old male who developed a post-traumatic subdural abscess which was followed by meningitis. (NP 182)

PATHOLOGY: The leptomeninges are the seat of a thick fibrino-purulent exudate, with polymorphonuclear leukocytes predominating (A). In the upper cortical layers the ganglion cells show marked degenerative changes and there is recent proliferation of astrocytes and microglia. The cortical vessels have somewhat hyalinized walls as well as a swollen endothelial lining. Here and there polymorphonuclear leukocytes have penetrated into the cortical perivascular spaces (B) a feature which warrants the term "encephalitis".

Reference: Courville, C. B.: Pathology of the central nervous system, Mountain View, California, Pacific Press Pub. Ass'n., 1937, pp. 112-116.



NEG. 72882a X 15 NEG. 72882b X 650

Slide 98

DISSEMINATED SCLEROSIS

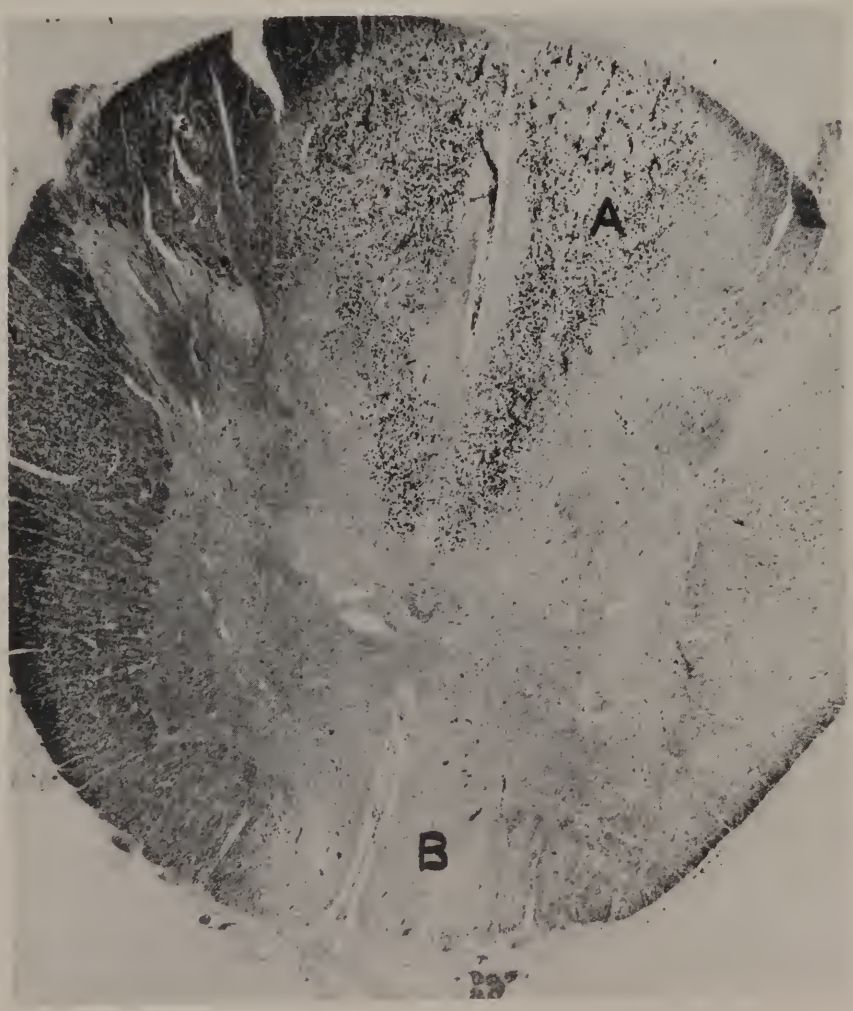
Fat Stain

CLINICAL NOTE: Not available.

PATHOLOGY: The demyelinated foci in the posterior columns are recent, as judged by the large numbers of fat-laden gitter cells (compound granular cells) therein (A). In this region some of the fat lies free in the tissue.

The anterior columns (B) are the seat of an older process. Here the few scavenger cells are present mainly in the vicinity of blood vessels, to which they have wandered from their initial position in the parenchyma. The anterior horn cells contain dense collections of fat granules.

Reference: Greenfield, J. G., and King, L. S.: Histopathology of the cerebral lesions in disseminated sclerosis, Brain 59: 445, 1936.



Slide 99 NEG. 72888 X 15